University of Alberta

Memory Deficits in Fetal Alcohol Spectrum Disorder

by

Jacqueline R. Pei ©

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the requirements for the degree of Doctor of Philosophy

Department of Educational Psychology

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UNIVERSITY OF ALBERTA

FACULTY OF GRADUATE STUDIES AND RESEARCH

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled *Memory Deficits in Fetal Alcohol Spectrum Disorder* submitted by Jacqueline Raylene Pei in partial fulfillment of the requirements for the degree of Doctor of Philosophy

Dr. Morris J. Cohen, External Examiner

Date: Sept.16,2003

Dedication

This project is dedicated to my children, Nathan, Jacob, and Sydney, for their constant reminder of what is really important.

Abstract

This dissertation consists of three separate papers on the assessment and diagnosis of Fetal Alcohol Spectrum Disorders (FASD). The first paper is a review paper in which the evolution of the diagnosis of FASD is discussed. The complex merger of medical and psychological bodies of research is described, as well as the unique difficulties confronting diagnosticians evaluating this syndrome. The article also presents an updated approach to diagnosing FASD. As such, the material presented in this paper will inform practitioners regarding the unique features in FASD, and begin the process of equiping them for work with this diverse population.

In the second paper an empirical study of the specific types of memory deficits present in children with FASD is presented. Specifically three areas of memory are examined (1) visual memory, (2) verbal memory, and (3) learning. Thirty children participated in the study (mean age 12-10) and were administered a comprehensive measure of memory functioning (The Children's Memory Scale; CMS) as well as a measure of cognitive functioning (Weschler Intelligence Scales for Children, Third Edition; WISC-III). Repeated measures indicated a main effect for differences in types of verbal memory as well as for learning verbal information. No independent significant effects were identified for visual memory or overall learning, nor for gender. Findings are discussed in light of Baddeley and Hitch's model of working memory.

In the third paper, an exploratory empirical study is presented investigating the relationship between deficits in three areas of functioning present in children with FASD. Thirty children (mean age 12-10) were administered a comprehensive measure of memory (CMS), cognitive functioning (WISC-III), and executive functioning (Children's Category Test; CCT). Analysis revealed low correlations between performance in most areas, indicating little relationship between the measures. Moderate correlations were observed for the WISC-III and the CCT and the visual and attention/concentration indexes of the CMS. Overall, tremendous variability both between and within the children was observed. As such, the unique requirements of assessment with this population are discussed.

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I. Introduction

Fetal Alcohol Spectrum Disorder (FASD) is a variable cluster of physical, neurological, cognitive, and behavioural characteristics. While this cluster tends to be different for different individuals, several areas of impairment appear fairly consistently among children who have been exposed to alcohol prenatally (e.g., Mattson, Riley, Gramling, Delis, & Jones, 1998; Olson, Feldman, Streissguth, Sampson, & Bookstein, 1998; Randall, Ekblad, & Anton, 1990). In the following dissertation these characteristics will be addressed in an effort to improve our ability to understand and diagnose this complex disorder. However, before looking at the research on FASD, it is important to understand why it is currently receiving so much attention by clinicians, researchers, and the general public.

According to a literature review by the Institute of Medicine (1996), Fetal Alcohol Syndrome (FAS) has prevalence rates ranging from as low as 0.2 per 1000 births to as high as 189 per 1000 births among Natives in northwestern Canada. In fact, although it is not always accompanied by mental retardation, FAS has been described as the leading identifiable cause of mental retardation resulting from teratogen exposure in the United States and Europe (Abel & Sokol, 1986, 1987). Abel and Sokol (1987) reviewed worldwide incidence rates and calculated that FAS was recognized in infants in prospective studies at a rate of 1.7 per 1000 live births, and as children were recognized at older ages this rate climbed to 3.3 per 1000. Niccols (1994) concludes that these figures signify that approximately 6500 new cases occur in the United States each year, a significant increase over Clarren's (1986) estimate of only 350 new cases of FAS per year, and which in turn may suggest that FAS has been grossly underdiagnosed and underreported. Recent research indicates that approximately one to three babies out of every 1000 live deliveries have FAS, and that when FAS and Alcohol Related Neurodevelopmental Disorder (ARND) groups are combined, that number grows to 9.1 children out of every 1000 (Sampson, Streissguth, Boodstein, & Barr, 2000). In Canada, the estimated incidence of FAS is between 0.5 and 3 per 1000 live births, and the estimated incidence of FASD is 10 per 1,000 live births (Alberta Medical Association, 1999a). While somewhat varied, these incidence rates still clearly indicate that there are a significant number of children being born each year with some degree of brain injury due to prenatal alcohol exposure.

As ongoing research continues to identify the broad range of problems that may be present in children exposed to alcohol in utero, including cognitive delays, memory deficits, and problems with social functioning, the need for accurate identification and appropriate intervention is apparent. In addition, many secondary effects of teratogen damage have also been identified in those diagnosed with FAS including mental health problems, expulsion from school, criminal activity, inappropriate sexual behaviour, and substance abuse problems (Fast, Conry, & Lock,1999; Streissguth, Barr, Kogan, & Bookstein, 1996). While it is difficult to prove causation in the role of FAS to secondary disabilities, there is certainly sufficient evidence to indicate a strong relationship between these two. Considering the possibility of secondary effects of FASD, then, it is clear that if, as practitioners, we are to best meet the needs of these children, accurate diagnosis is essential for the prevention of possible cumulative problems.

In addition to preparing practitioners for appropriate consideration of the needs of FASD children, accurate diagnosis also allows for identification of a birth mother. In this way support can be provided to the birth mother and her family to prevent future children from being affected, as the risk of recurrence of FAS in families with one affected child is 771 per 1000 live births (Alberta Medical Association, 1999b). In short, given the high cost of FASD and related secondary disabilities to the individual, their family, and society, further research in this area to better understand the implications of prenatal alcohol exposure is clearly warranted for both intervention and prevention purposes. With this goal in mind, the present research is designed to continue building on the current knowledge base in FASD and further our understanding of this disorder.

This dissertation consists of three separate papers, each dealing with the challenge of diagnosis of Fetal Alcohol Spectrum Disorders. In chapter II the evolution of FASD diagnosis since it was first described in 1973 is examined. As researchers and clinicians jointly continue to decipher the deficits that accompany children with FASD, the diagnostic process has had to change. However, keeping up with the current practice is challenging. In an effort to remediate this knowledge gap, the first paper briefly presents the history and development of diagnostic criteria for FASD and based on a comprehensive review of the literature emphasizes best practices in the field. In chapter III the specific types of memory deficits seen in children with FASD diagnoses are examined from the perspective of a model of working memory. Specifically, differences between visual and spatial, visual and verbal, and delayed and immediate memory functioning in children exposed to alcohol prenatally were investigated. In chapter IV the focus was on the relationship between three different measures when used to evaluate the level of functioning in children with FASD diagnoses. In particular, memory, cognitive ability, and executive functioning were compared to determine whether performance on a memory measure would predict performance on a measure of executive functioning. In addition, conclusions are drawn regarding unique considerations that must be kept in mind when conducting assessments with this population.

In all papers the focus was on increasing knowledge in the areas of FASD so that those working with this population can make accurate diagnoses and subsequently implement focused interventions. In the first paper, this is accomplished through a comprehensive review of the diagnostic changes in FASD, with an emphasis on the current diagnostic practices that have been designed for this unique population. In the second paper, specific memory deficits for children with FASD are examined and interpreted in the light of a specific working memory model. Finally in the third paper, the relationship between the broad constructs of executive functioning and memory are explored in search of evidence that deficits in both areas may be traced to a common underlying mechanism.

Diagnosis in FASD

In chapter II a review of the evolution of diagnostic criteria for FASD is presented. This paper contributes to the FAS literature by providing professionals who encounter children with FASD with an understanding of the diagnostic complexity of FASD. The review begins with the history of the term FAS, from which the name Fetal Alcohol Syndrome (FAS) was applied to describe the pattern of anomalies in children born to alcoholic mothers. The three criteria for making a diagnosis are also presented and a discussion of the evolution and refinement of the criteria is provided. Next the process of confirming the teratogenicity of alcohol is briefly introduced, leading to the conclusion that alcohol is clearly a teratogen (e.g. Randall, Ekblad, and Anton, 1990).

Having established the teratogenic property of alcohol, the research that ensued in an effort to better understand this syndrome is reviewed. Detailed descriptions of physiology and neurology as well as functional deficits are presented. At the same time the wide range of variability present in this syndrome is underscored. Consequently, the need for clarity and consistency is a recurring theme. At the same time, however, the awareness of the wide range and variety of deficits makes consistency a difficult goal. A crucial acknowledgement in the field is that a child can be alcohol effected and yet not present with the physical symptomology. Consequently, various diagnostic labels such as Fetal Alcohol Effects (FAE) and Prenatal Alcohol Exposure (PAE) began to emerge. In turn, with the absence of physical indicators, diagnosis became even more uncertain. To address this researchers began to investigate the cognitive and behavioural deficits present in FASD as a consequence of the brain injury incurred in utero (e.g. Abel & Sokol, 1986). This period of time, in which large amounts of research was generated in an effort to better understand FASD, has been referred to as the Gestalt period of diagnosis. During this period, FASD was considered in holistic terms and no clear diagnostic criteria existed. While study into the deficits present was moving ahead, translating this information into practical, clinical forums moved slowly. Eventually the Institute of Medicine (1996) attempted to provide more diagnostic structure and consistency through the description of five diagnostic categories. However, the general approach to diagnosis remained the same.

Problems with the diagnostic process are also explored. In particular, in the first paper the unique features of FASD were investigated, and the necessity for different disciplines to work together to fully capture the complexity of the disorder is discussed. Overall, the argument presented is that the nonspecific nature of the Gestalt approach contributed to a large amount of misdiagnosis, and that consequently misconceptions and misunderstanding about FASD continued to proliferate.

Fortunately, the diagnostic evolution did not end at this point and in paper #1 the most recent changes are described. Unlike previous changes to diagnosis, current update has presented a different system altogether. Introduced as the 4-Digit Diagnostic Code for FASD, this system embraces the multi-disciplinary nature of FASD as well as the wide range of variability. It uses a team approach to diagnosis, and documents the magnitude of expression of the four key components of the syndrome: 1) growth impairment, 2) facial phenotype, 3) evidence of brain injury, and 4) prenatal alcohol exposure. This approach seems not only to provide more specificity in diagnosis, but also permits individualization of intervention as necessary. Unfortunately, it also requires the specialized training of a team to implement this approach appropriately. As such, it is still a long way from universal implementation, and many practitioners remain unaware of this approach. Consequently, this paper concludes with a call for communication and education for clinicians and researchers regarding diagnosis of FASD as without accurate diagnosis appropriate intervention and prevention cannot be implemented. In helping to establish the necessary clarity and consistency in diagnosis, the four-digit system of diagnosis has been implemented in sample selection for the following empirical papers.

Memory in FASD

In the second paper of this dissertation the need for a clearer clinical picture of FASD is addressed. Whereas the first study is essentially a review article of the diagnostic debate in FASD, the second study is empirical and utilizes quantitative research methods in an effort to better understand the nature of the deficits in FASD. In particular, the focus of this study was the types of memory deficits present in children with FASD. As such, three questions specific to memory functioning in children with FASD were examined: (1) Is spatial memory more impaired than facial recognition memory? (visual memory), (2) Is memory for stories more impaired than memory for words? (verbal memory), and (3) Can memory deficits in FASD be attributed primarily to learning or encoding deficits rather than retrieval deficits?

In order to answer these main questions a comprehensive model of working memory is introduced as a framework through which memory in FASD is then examined. In this model, first introduced in 1974 by Baddeley and Hitch, working memory is divided into several functional components, making it a useful clinical model. In the present study, evaluation of memory within this theoretical framework is accomplished through administration of a comprehensive memory measure, the Children's Memory Scale (Cohen, 1997).

After establishing a theoretical framework within which memory can be explored, research specific to memory deficits in FASD is reviewed. In this information, specific deficits in spatial memory functioning are described, while memory for objects is preserved (Uecker & Nadel, 1998). The second question concerns the acquisition of verbal information, in which deficits have been described in children with FASD, though comparison between types of verbal memory has been sparse. The last question addressed in Chapter III relates to recent conclusions within the FASD literature, that the true source of memory deficits in FASD lies in the process of encoding or learning the information (Mattson & Roebuck, 2002).

Assessment of FASD

The third and final paper of the dissertation focuses on the assessment of deficits in children with FASD. In Chapter IV, I describe the types of deficits identified for children with FASD in the domains of intelligence, executive functioning, and memory, and the unsuccessful search for a pattern between these areas. While some systems are identified as potentially more vulnerable to alcohol exposure, a high degree of variability is apparent between and within FASD children. Despite this diversity, the possibility that an underlying mechanism may be responsible for deficits observed in memory and executive functioning has been proposed (Kodituwakku, et al. 1995). The present research is the first study to directly explore the relationship between executive functioning and memory using a comprehensive clinical measure of each, specifically the Children's Category Test and the Children's Memory Scale. The Weschler Intelligence Scale for Children, 3rd edition, was also administered to rule out the possibility that any results would be affected by cognitive functioning.

This dissertation concludes in Chapter V with a discussion of the main findings and limitations of the presented studies, as well as educational implications and some ideas for future research.

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II. A Review of the Evolution of Diagnostic Practices for Fetal Alcohol Spectrum Disorder

Introduction

Fetal Alcohol Spectrum Disorders (FASD) are described as the leading causes of preventable emotional, behavioural, cognitive, and social dysfunction (Abel & Sokol, 1986, 1987; Streissguth, 1997; Streissguth & O'Malley, 2000). Until recently, however, FASD has received relatively little recognition, and, in fact, has been described as one of the most under-treated of the lifelong developmental disabilities within the mental health disciplines (Streissguth & O'Malley, 2000). Spurred on by recent high profile reports of crimes committed against or by those who have been diagnosed with FASD, discussion of this syndrome has spanned several disciplines and research has flourished. Teachers, social workers, psychologists, and physicians are just a few of the professionals finding that knowledge and understanding of FASD would be an asset to their work.

Fortunately, there are positive outcomes for prenatally exposed children who are diagnosed and treated early (Streissguth, 1997), and thus the need and urgency for global understanding and consistent diagnostic and assessment approaches grows. Accurate assessment is crucial for appropriate diagnosis and intervention. In fact, Whaley et al. (2001) argue that early intervention based on accurate diagnoses "could prevent the social skills deficits evidenced in these children as they grow older. For this reason, there is a need for clinicians to screen patients for prenatal alcohol exposure when making decisions about treatment options" (p. 1022). Moreover, Clarren and colleagues (2000) emphasize the importance of accurately determining etiology of observed behaviour for two key reasons. First, they state that etiological awareness alerts clinicians to the complex nature of the profile, and allows for an attitude change from seeing a child as disobedient to seeing a child as disabled. Secondly, they argue that accurate identification may help prevent prenatal alcohol exposure in the biological mother's future pregnancies, as she will be identified and may receive necessary support within a prevention program. As such, appropriate diagnosis is essential, so that effective intervention may follow. Unfortunately, three key problems plague the FASD field: 1) problems with diagnostic consistency, 2) knowledge of FASD and appropriate interventions for FASD, and 3) public policy and funding initiatives that fail to effectively address the diverse needs of this population.

As with many disorders, current knowledge about FASD is tautological. Because there is no definitive test for FASD, researchers and clinicians diagnose FASD symptomatically and then look back to these abnormalities to better refine diagnosis. Consequently, our knowledge base continues to grow and evolve and it is difficult for practitioners to remain abreast of all developments. Thus, the purpose of this review is to recap the changes in diagnosis of FASD over the last thirty years, provide clarity into the changing terminology, identify some of the unique difficulties confronting diagnosis of FASD, and, finally, to describe the most current diagnostic model being used in FASD and best practices that can be applied in working with this population.

The Early Years

As with any disorder, definitional issues are at the heart of diagnosis. The features of Fetal Alcohol Syndrome (FAS), as it was first named, were originally described in 1968 by Lemoine et al., who identified a consistent set of physical features in infants of mothers with alcoholism. Then in 1973, Kenneth Lyons

Jones and David W. Smith, dysmorphologists at the University of Washington Medical School, identified a "similar pattern of craniofacial, limb, and cardiovascular defects associated with prenatal onset growth deficiency and developmental delay" in eight unrelated children of three ethnic groups born to alcoholic mothers (Jones, et al., 1973, p. 1267). The authors thought the prenatal alcohol exposure might be responsible for some of the functional abnormalities and joint malpositions comprising this syndrome. In a separate paper, Jones and Smith (1973) named this pattern or cluster of anomalies in children born to alcoholic mothers Fetal Alcohol Syndrome (FAS) and identified diagnostic criteria based on three features: 1) pre- and/or postnatal growth deficiency; 2) a distinct pattern of craniofacial malformations; and 3) central nervous system (CNS) dysfunction.

Diagnostic development did not move forward directly from this point. Instead, despite the clinical evidence, there was a resistance within the scientific and clinical community to the idea of alcohol as a teratogen (Randall, 2001). Consequently, the next step required providing the necessary evidence to confirm the teratogenicity of alcohol, and to better understand the effects of alcohol in utero. Animal studies in particular in this area allowed researchers to confirm that alcohol is, on its own, clearly a teratogen (Chernoff, 1977; Randall, 1987; Sulik, Johnson, Webb, 1981). In such studies on experimental animals, potentially confounding effects, such as nutrition, other drug exposures, and postnatal rearing conditions, were controlled to help rule out alternative explanations and provide strong support for the argument that alcohol itself is a teratogen. In fact, prenatal alcohol exposure has been demonstrated in many species to cause all four teratogenic endpoints (death, malformations, growth deficiency, and functional deficits) depending on the dose, timing, and conditions of exposure (Randall, Ekblad, & Anton, 1990; Schenker, et al., 1990; West, 1986). As such the teratogenicity of alcohol was firmly established.

As animal studies began to establish credibility for the conclusion that alcohol is a teratogen, researchers worked to better define the three diagnostic criteria for FAS as first described in 1973. The first criteria, necessary for a diagnosis of FAS, are the facial features as this is the only component of FAS that cannot be explained by any other disorder. No one can receive a diagnosis of FAS without the presence of these characteristic facial features, which, when taken as a whole, are described as the "FAS face." The anomalies that appear to create the FAS appearance are localized to the central facial region and form a sort of "T" (Institute of Medicine, 1996). The upper horizontal bar of the T is formed by the eyes and inner canthal region. In this area, the palpebral fissures (eye slits) are short, while the inner canthal distance is more normal. Ptosis, high lateral arched eyebrows, and epicanthal folds are associated anomalies that may be present but do not define the horizontal bar without the shortened palpebral fissures. The vertical bar of the T is defined by the nose and philtral region. In this area, the philtral furrows are flattened and the upper lip is thinned. While precise assessment of these characteristics through consistent interpretation of measurements has yet to be universally implemented, the issue has been addressed by researchers, such as Astley and Clarren (1995, 1997), who have developed criteria for the facial phenotype of FASD. Diagnosis using these criteria, however, becomes more difficult when the individual ages, as the facial malformations often disappear or become less pronounced in adulthood (Conner, et al., 1999). Moreover, while the "FAS face" helps to establish the presence of FASD, absence of the face does not rule out FASD (Mattson et al., 1998).

The second criteria, evidence of growth retardation, must be present in at least one of the following areas: low birth weight for gestational age, decelerating weight over time not due to nutrition, and disproportional low weight to height compared to medical norms (Institute of Medicine, 1996). However, as with the facial phenotype, this feature is not a necessary component as the unique FASD facial characteristics as well as brain dysfunction have been described in the absence of any growth deficiencies (Institute of Medicine).

The final criterium for a diagnosis of FAS is Central Nervous System (CNS) dysfunction. Research has revealed that there is no question that prenatal alcohol exposure affects the developing brain (Mattson & Riley, 1998). As early as 1973, at which time the first brain of an FAS infant was studied in autopsy, clear structural abnormalities were observed by researchers (Jones & Smith, 1973). The causal relationship between prenatal alcohol exposure and adverse developmental outcome has also been clearly established (Streissguth, 1997), though it is the specific CNS deficits that are more difficult to pinpoint. A consistent pattern of brain anomalies has yet to be confirmed, and some researchers have even questioned whether a specific pattern of impairment even exists (Clarren, 1986; Peiffer, et al., 1979). However, current medical techniques (e.g. MRI) that allow for a more systematic measure of alcohol's teratogenicity have provided support for the possibility that some patterns of deficits may be present due to the varying levels of susceptibility to the effects of alcohol during development (e.g. Mattson et al., 1992; Riley et al., 1995; Roebuck et al., 2002). Other factors, such as the developmental period during which exposure occurs, the dose, and protective features in the mother have been linked to CNS vulnerability. As such, the existence of a "sensitive period" of development may exist in which the toxic effects of alcohol would have the most profound effects, or even specific and consistent effects, on the developing brain, though competing external factors make this difficult to determine.

Studies in FASD have begun to reveal the ways in which the brain is affected (e.g. Roebuck, Mattson, & Riley, 2002). Specifically, alcohol-induced defects in neuronal migration, reduced number of neurons in the mature cortex with the same origin, delay in cortical neurons being generated, and alteration in the distribution of neurons on a specific day have been demonstrated to produce changes in the developing brain (Miller, 1986, 1988, 1993). In addition, researchers have examined the brain and identified several areas that tend to be more susceptible to the effects of alcohol, including reduced size of the basal ganglia, corpus callosum, and diencephalon (Clarren, 1986; Mattson & Jernigan, 1994; Mattson et al., 1992; Riley et al.,1995; Sowell et al., 1996). As research into structural deficits continues, it is complemented by neuropsychological evaluation that provides insight into the possible consequences of such structural damage, the functional deficits present in children with FASD. In other words, these changes to the brain are of great importance in the etiology of CNS dysfunction which includes impairment in language, verbal learning, memory, academic skills, fine-motor speed, and visual-motor integration (Mattson & Riley, 2000).

As knowledge about prenatal alcohol exposure began to grow, other diagnoses parallel to FAS emerged to account for effects that may have some but not all of the characteristics of FAS. Since the CNS develops during the entire pregnancy, as opposed to the craniofacial features that only develop between the nineteenth and twenty-second days of pregnancy, the teratogenic effects of alcohol can affect the CNS without being evident in the facial features. Thus, researchers have suggested that children without the characteristic facial features of FAS, but who have been exposed to alcohol prenatally, may still have incurred damage to the brain which could be evident in their significant cognitive deficits. Mattson and colleagues (1998) indicate that "children with histories of heavy prenatal alcohol exposure display neuropsychological deficits and furthermore, that these deficits persist in the absence of the pattern of physical features associated with FAS" (p. 152). Both animal and human studies have revealed that hyperactivity, problems with response inhibition, attention deficits, poor habituation, poor coordination, and poor state regulation is associated with alcohol use during pregnancy (Mattson & Riley, 1988), and many of these deficits have also been found in children who do not necessarily meet the criteria for a diagnosis of FAS (Brown et al., 1991; Coles, et al. 1997; Goldschmidt et al., 1996; Jacobson et al., 1993). In general, accuracy and precision in diagnosis could not be assured because of the lack of diagnostic specificity (Astley & Clarren, 2000). Consequently, researchers and clinicians were forced to re-examine the original diagnostic criteria.

The Institute of Medicine's (IOM; 1996)) report on FAS addressed the lack of diagnostic clarity by identifying five categories of alcohol-related disability. The first category is FAS with confirmed maternal alcohol exposure, which consists of facial dysmorphology, growth retardation, and structural brain anomalies. The second category is FAS without confirmed maternal alcohol exposure, which includes the same deficits as category one but without the confirmed alcohol history. The third category is partial FAS, with confirmed maternal alcohol exposure in which there may be some facial dysmorphology, some growth retardation, and some CNS dysfunction, as well as behavioural or cognitive abnormalities. The fourth category is Alcohol-Related Birth Defects (ARBD) which describes the presence of any physiological features. And the fifth category is Alcohol-Related Neurodevelopmental Disorder (ARND). The ARND criteria include structural brain anomalies, decreased cranial size at birth, neurological hard and soft signs (such as impaired fine motor skills, neurosensory hearing loss, poor tandem gait, and poor hand-eye coordination), and/or evidence of a complex pattern of behaviour or cognitive abnormalities that are inconsistent with developmental level and cannot be explained by familial background or environment alone. These disorders which do not fullfill the full FAS criteria have also been referred to in the literature as Fetal Alcohol Effects (FAE) and Prenatal Alcohol Exposure (PAE). Within the IOM framework, the latter categories are believed to be a less severe version of FAS due to the absence or reduction in facial dysmorphology and other physical features as well as fewer cognitive deficits, although behaviourally both disorders are marked by increased impulsivity, aggression, and social problems (Jacobson & Jacobson, 1999). This diagnostic approach has been described as a Gestalt approach to diagnosis as it entails a look at the whole clinical picture to form a judgment regarding whether the pattern that presents is consistent with a diagnosis of FAS or ARND.

Problems with the Gestalt Approach

The gestalt approach, even after being enhanced by the IOM criteria, has still been plagued by criticism for failing to establish a means for providing valid and reliable diagnoses. While more detail was provided, problems remained. Two areas in particular emerged as problematic: 1) the multidisciplinary nature of FASD diagnosis, and 2) the complexity of FASD.

A Multidisciplinary Diagnosis

The first area of concern relates to the variety of disciplines that play a role in diagnosis, but each of which in and of themselves lack the full range of expertise required to provide a comprehensive diagnosis. For instance, the Institute of Medicine (1996) stated in their publication that while a trained clinician (such as a psychologist) may diagnose ARND for the purposes of screening and referral, the medical diagnosis of FAS must be left to dysmorphologists and clinical geneticists. This separation of the two presentations of prenatal alcohol exposure, while appropriate, mimics to some degree a division within the literature. While the medical researchers explore the medical abnormalities present in children prenatally exposed to alcohol, the psychological community is focusing their research on the neurological damage incurred through early alcohol exposure, and subsequent cognitive and behavioural impairment. This division, while useful in research, creates some practical concerns for diagnosis, and day-to-day work, because the combination of both medical and psychological elements may not exist naturally in many current clinical settings. Consequently, FAS is vastly misdiagnosed (Codero, et al., 1994). Contributing to this problem has been the lack of universal standards for diagnosis. While some areas offer training for physicians, and provide guidelines for best practices, they are not required to receive this training and many remain uninformed on this issue. Similarly for psychologists, while training exists, no universal criteria for diagnosis have been accepted. As will be discussed shortly, some steps are being taken to rectify this absence of clearly defined standards, but to date, use of this new material is far from universal and remains unknown to many.

The standards problem is combined with problems within the professions involved in diagnosis. Susan Astley and colleagues (2000) suggest three issues have contributed to the failure to diagnose FAS medically: 1) quite simply, some physicians may have no knowledge of the existence of FAS or how to diagnose it, 2) physicians may fail to recognize the unique and complex presentation of FAS, resulting in a symptomatic but ultimately inaccurate diagnosis such as ADHD and implementation of programs geared for these disorders, 3) diagnoses and intervention with FAS children requires coordination of many services and agencies, a role that she suggests physicians usually have neither the training nor the networks to play. Because of these three problem areas, Astley says that physicians fail to perceive the benefits of diagnosis and therefore tend to underdiagnose FAS. A similar analysis could be made of psychologists with regards to consideration of CNS dysfunction, as it is likely that comparable problems exist for this profession when it comes to making a diagnosis. Overall, then, the interdisciplinary nature of FASD, and the tendency of many clinicians to operate independently, may create difficulties with knowledge of FASD, appreciation of the importance of diagnosis, as well as the necessary networks to communicate effectively with one another regarding both diagnosis as well as follow-up.

The picture can become even more complicated and divided when the possibility of even more practitioners, all potentially operating in isolation, become involved. It appears as though CNS deficits are at the core of the involvement of other professionals in educational, mental health, and/or social welfare professions. Streissguth and colleagues (1996) have identified the consequences of many of these deficits as Secondary Disabilities. In particular, they have reported that of those diagnosed with FAS, over 90% have mental health problems, 60% have been expelled from school, 60% have been in trouble with the law, 50% have been or are in jail, 50% have engaged in inappropriate sexual activity, and 30% have alcohol or drug problems (Streissguth, et al.). Further support is provided in research by Fast, Conry, and Lock (1999) in which they found that found that children with FAS were disproportionately represented in the juvenile justice system. Clearly, the severity of these problems will require the involvement of many disciplines to effectively identify the needs of each child. But, with so many professionals involved, an accurate diagnosis can be difficult to establish without effective communication networks and a comprehensive understanding of FASD by all parties involved.

A Complex Diagnosis

The second area of difficulty involves the complexity in making a diagnosis. Given the diverse features present in FASD, in addition to often dubious maternal reports of alcohol consumption and lack of familiarity with the disorder (Ernhart et al. 1995), accurate diagnosis is frequently identified as a concern. Unfortunately, the Central Nervous System (CNS) dysfunction associated with FAS, while more prevalent as a prenatal alcohol effect, has not been considered to be as specific or unique as the facial dysmorphology, thus making diagnosis more dependent on the Gestalt approach. As noted earlier, this dysfunction is neurological in origin and spans all areas of cognitive and behavioural functioning, resulting in the dysfunction presenting differently in different individuals depending on factors such as the timing or extent of alcohol exposure. The Institute of Medicine (1996) states that these problems include disordered motor development, diminished intellectual functioning, delayed and disordered speech and language development, problems in social perception, memory deficits, and deficits in response inhibition and attention. Consequently, knowledge of the entire spectrum of impairment is necessary. Even then, accurate diagnosis can be elusive since psychologists might still diagnose symptoms without truly understanding etiology.

While researchers and clinicians have studied FASD in an effort to identify a consistent pattern of deficits, instead, what they have found for the most part in many areas is a lack of consistent deficits. As yet, no single type of CNS damage or pattern of dysfunction has been identified that characterizes all children who have been prenatally affected by alcohol (Streissguth, 1997). Research examining developmental outcomes has yielded variable results (Institute of Medicine, 1996) that could have many origins - different patterns of maternal alcohol consumption and different levels of individual susceptibility are just two possible factors. At the same time it may be more likely that general trends could be identified if all studies were identifying FASD using consistent diagnostic criteria. This is particularly true in the case of ARND where the more objective physical characteristics are absent. Thus, there are concerns that ARND is either being over- or under-diagnosed, which poses a considerable hurdle for researchers. Furthermore, the population varies within the research—some focus on FAS, some on ARND and some mix the groups. As such the Gestalt approach has had its drawbacks as identification is nonspecific, and, with so much variability, misconceptions abound. While this is a reality for this disorder, it certainly seems that achieving increased consensus and clarity would not only assist with diagnosis and research but also with the vital interventions that follow.

Best Practices and The Four Digit Code

In 1997, Astley and Clarren introduced the 4-Digit Diagnostic Code for diagnosis of Fetal Alcohol Spectrum Disorders (FASD). This new method was created partly in response to the Institute of Medicine's recommendations that a more reliable and valid set of diagnostic definitions be adopted. Accordingly, this system is designed to determine the degree to which the facial dysmorphic features, growth retardation, and CNS involvement exists as a consequence of prenatal alcohol exposure, rather than defining severity. An added advantage to this system is that it uses a team approach that allows for a comprehensive review of function in all areas, thus ensuring communication between medical and psychological diagnosticians, as well as other key mental health professionals. Briefly, this system documents the magnitude of expression of the four key components of the syndrome, specifically: 1) growth impairment, 2) the FAS facial phenotype, 3) evidence of brain damage, and 4) prenatal alcohol exposure, on separate four-point Likert Scales (Astley & Clarren, 2000). A rank of "1" on any scale means a finding within the normal range. A "4" on any scale indicates a finding that corresponds with accepted cases of FAS. A score of "2" or "3" specifically defines intermediate steps between typical and atypical presentation of FAS characteristics. These scales do not necessarily measure increasing severity, rather they are scales of greater confidence that FAS is present, and as such, a diagnosis of FAS requires ranks of 3 or 4 in all categories. There are, however, many other possible alcohol-related diagnoses provided depending on the code obtained, in which case there is much more room for consideration of the entire spectrum of this disorder. With this in mind, the most recent term to be used has been Fetal Alcohol Spectrum Disorders (FASD) as it allows for the continuum of deficits to be considered for discussion and research purposes. This said, categorical terms such as FAS and ARND, which establish artificial boundaries within the spectrum, are still required at this time as they provide clear diagnostic terms that permit access to funding and supports.

Into the Future

Diagnosis of FASD has evolved greatly over the last thirty years, from its original description in 1973 based on medical observation, to its current form in which diagnostic criteria spans the domains of medical, psychological, and educational functioning. Current thought reflects the knowledge that FASD is not a dichotomous condition. Rather its clinical features and even the history of alcohol exposure itself range along separate continuums from normal to clearly impaired. As a result clinicians and researchers have been challenged with the task of diagnostic consistency. Nonetheless, despite the variability within this population in terms of specific deficits, there is still a consistent pattern in the presentation of these deficits that includes increased social problems, aggression, inattention, and delinquency (Mattson & Riley, 2000). In addition, this impairment has been identified in many areas of functioning, regardless of whether or not the full criteria for FAS are met. As well, we are beginning to understand that FASD can affect individuals in different ways. In other words, while many of the secondary disabilities, or consequences, of the deficits may be similar, the path to these disabilities may be varied. The apparent incongruity will likely continue to be a focus of research as we continue to seek a clear understanding of diagnoses of FASD, and subsequently implement interventions and apply appropriate policy. It seems likely that the diagnosis of FASD will never be as simple as a single test or characteristic. Instead, children and adults will need to be considered on an individual basis to establish their specific needs, as reflected in the 4-Digit Code style of diagnosis. However, in order to see this implemented, clinicians and researchers need to be informed about its existence as well as its importance in the diagnostic process, both for intervention and prevention purposes. As long as clinicians continue to make diagnoses or researchers continue to conduct studies based on outdated criteria, misdiagnosis will likely ensue.

Finally, policymakers will need to recognize the extent of the deficits present in FASD and provide the financial support necessary for the individualized intervention that is required. This is not a hopeless problem without solution, rather one that requires special consideration and attention to its unique and varied presentation. Only through a greater understanding of the needs of children, adolescents, and adults with FASD will we be able to activate and implement the resources that will make a difference in the lives of all of the individuals involved.

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III. Patterns of Acquisition and Retention of Verbal and Nonverbal Information in Children with Fetal Alcohol Spectrum Disorders

Introduction

Fetal Alcohol Spectrum Disorders (FASD) often include many indicators of Central Nervous System (CNS) dysfunction. Unlike the physical features, such as facial dysmorphology and growth retardation, a consistent pattern of CNS dysfunction has been more difficult to describe. Research into FASD has identified impairment in many areas of functioning including delayed motor development (Barr, Streissguth, Darby, & Sampson, 1990), cognitive impairment and learning difficulties (Conry, 1990; Streissguth, Barr, & Sampson, 1990), deficits in communication, daily living skills, social behaviour (Whaley, O'Connor, & Gunderson, 2001), memory deficits (Institute of Medicine, 1996; Mattson & Riley, 1998; Mattson & Roebuck, 2002), and response inhibition and attention (Nanson & Hiscock, 1990; Streissguth et al., 1994). A review of neurobehavioural deficits present in FASD was completed in 1998 by Mattson and Riley in which they concluded that "FAS is a devastating developmental disorder that is associated with a wide variety of neurobehavioural deficits… in language, motor, learning, and visuospatial functioning."(p.291). However, despite this growing understanding of the breadth of possible impairment in children with FASD, no clear pattern of neurobehavioral deficits has yet been identified. Consequently, diagnosis is difficult and many children and adolescents are not appropriately diagnosed and thus any dysfunctional behaviour they may exhibit is often misunderstood. Due to a failure to understand that their unpredictable behaviour stems from organic brain damage, clinicians run the risk of exacerbating problems they are attempting to ameliorate, through their intervention efforts (Streissguth, 1999). Only once a child's behavioral and/or cognitive deficits are fully understood is effective action possible.

One way to improve diagnostic accuracy is to continue to develop a clear clinical picture of children with FASD. This can be accomplished through close examination of specific deficits described in FASD. Two such areas where deficits may exist are memory and learning. Memory and learning deficits, in children with FASD in particular, are extremely problematic because of the effect they have on many other skills and abilities. Language acquisition (e.g. Adams & Gathercole, 1995), academic performance (e.g. Schunk, 1996), and even competent daily functioning have memory and learning functions at their foundation (e.g., Lezak, 1995). Thus, to understand these deficits is to better understand their consequences and impact on children with FASD. And, in turn, intervention can be more specifically tailored to the needs of these children in both educational and home settings.

But first, in order to appreciate the types of memory deficits evident in children with FASD, it is necessary to explore some basic concepts of memory. In so doing, it will then be possible to discuss the specific memory deficits present in children with FASD from within a theoretical framework of memory and learning. The model of working memory presented by Baddeley and Hitch (1974) is a comprehensive model in which several components of working memory are described. By identifying different components, or systems, Baddeley and Hitch allow for investigation into the specific memory deficits described in FASD from a theoretical perspective that emphasizes function rather than simply storage capacity (Baddeley, 2002). In this way different types of memory functioning can be considered independently, which in turn allows for evaluation of the distinct effects different systems of memory may have on behaviour and learning. Thus, this functional model was used in the present study as a framework to address three questions of specific memory factors in children with FASD including: (1) is there a difference between spatial and object visual memory abilities? (2) is there a difference between two different types of verbal memory abilities? and (3) is the[illegible]ence between the children's ability to learn information and their ability to recall and retrieve it from long-term memory? By studying these areas of memory it is expected that specific areas of strength and weakness will be identified, which in turn will facilitate accurate diagnosis, and eventually lead to appropriate treatment, intervention, and planning.

We begin with an introduction to memory theory and a description of the specific memory model by Baddeley and Hitch. This will be followed by a review of the memory literature specific to children with FASD and a discussion of the current research. Finally, the results and discussion of the present research on the specific memory factors in children with FASD within this larger context will be presented.

Memory Theory

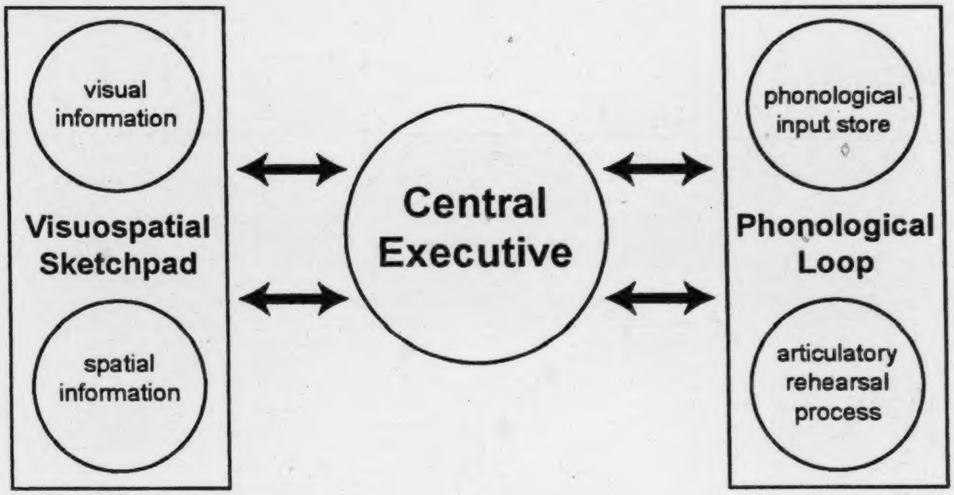
Memory is a dual system in which two distinct components co-exist: procedural memory and declarative memory. Procedural memory refers to those memories acquired without awareness or deliberate effort, and is not the focus of the current research. Declarative memory has been the focus of most memory research, and focuses on our ability to learn about and remember information, objects, and events (Lezak, 1995). It is this form of memory that is typically referred to in educational settings and tends to be the more common designation of memory.

Declarative memory, however, is not a unitary entity but rather a mix of complex functions that continues to be defined. The concept that memory could be fragmented into subcomponents was first proposed by William James in 1890, and reiterated almost 60 years later by Donald Hebb in 1949, although it was not until the late 1960s that experimental evidence of the fractionation of human memory began to develop (Baddeley, 1999). Then in 1968, Atkinson and Shiffron organized these proposed complex functions of memory into a modal model that consisted of a three-step process involving the sensory registers, short-term memory, and long-term memory. Since then more detailed models have been presented, generated by research evidence indicating that memory is even more complex than originally believed. One such model was proposed in 1974 by Baddeley and Hitch, and focused on short-term memory, or what they described as working memory. Baddeley explains that working memory is a very important component of human behaviour as it involves everything from mental arithmetic to reasoning and problem-solving. As such, working memory plays a significant role in both cognition and behaviour. To some degree, aspects of cognition and behaviour can be considered overt representations of our working memory processes. Using this framework of memory, then, with a population of children described as having cognitive and behavioural problems seems fitting.

Working Memory

Baddeley and Hitch (1974) proposed that memory consists of two memory stores regulated by a central executive. The central executive's purpose is to allocate incoming information to one of the two storage subsystems as well as to coordinate, monitor, and make use of the information in the subsystems (Figure 2-1).

Figure 2-1 Baddeley and Hitch's Working Memory Model



The first subsystem, or slave system as they called it, is called the phonological loop and consists of a memory store for the storage and manipulation of verbal information. The second subsystem is a memory store for the storage and manipulation of visuospatial information and is called the visuospatial sketchpad or scratch pad. The separation of these systems has been supported by research in which these verbal and visual working memory subsystems have been shown to be different (Baddeley 1986, 1992; Jonides et al., 1996). Furthermore, both subsystems have been divided to account for different functions. The visuospatial sketchpad has been divided into two separate subsystems, one for visual information and one for spatial information. This modification is consistent with research that has demonstrated the differences between visual and spatial processing systems in humans (Farah, Hammand, Levine, & Calvanio, 1988; Tresch, Sinnmon & Seaman, 1993). The phonological loop component is divided into the phonological input store and the articulatory rehearsal process (Baddeley, 1994). Baddeley assumed that the phonological loop holds speech-based information for a brief period (about 1.5 to 2 secs). The articulatory rehearsal process refreshes information in the passive store, if activated, and in this way can offset information decay. A closer look at these three systems illustrates the functional implications of each area.

Phonological Loop. This model of working memory has emerged frequently in the literature on memory and children. The phonological component of short-term memory has been closely linked with aspects of language development, including vocabulary acquisition (Gathercole & Baddeley, 1989; Gathercole & Pickering, 2000), speech production (Adams & Gathercole, 1995; Speidel & Nelson, 1989) and spoken language comprehension (Crain, Shankweiler, Macaruso, & Ben-Shalom, 1990). These researchers suggest that disruptions in phonological loop function (i.e., reduced phonological storage capacity and/or inefficient rehearsal abilities) lead to compromised comprehension, among other things, because insufficient amounts of incoming information can be immediately and readily retained in the phonological store for it to be processed. To test this, Montgomery (1995) examined the relationship between phonological working memory capacity and sentence comprehension in a group of school-age children with specific language impairment (SLI) and a group of younger children matched for receptive syntax. The children completed a nonsense word repetition task (testing phonological working memory capacity) and a sentence comprehension task. Results revealed that the children with SLI understood fewer longer sentences than shorter sentences as did their language-matched peers. The control children understood a comparable number of long and short sentences. In addition, a positive relationship was found between performance on the nonsense-word repetition task and the sentence comprehension task. Montgomery interpreted these findings to suggest that the limited phonological working memory capacity of the children with SLI compromised their sentence comprehension efforts. Because of their capacity limitation, these children were not able to store as much information at any given moment during processing, thus hindering their ability to construct a full sentence interpretation. In a companion study, Montgomery (2000) replicated these findings.

Findings from Montgomery's work provide support for Baddeley and Hitch's model of working memory. Moreover, Montgomery's research demonstrates the role that memory plays in a very important cognitive process—that of language comprehension. This is especially important when you consider the role of language and comprehension in both behaviour and cognition. Baddeley (1999) notes that this may be a significant problem for children. He states that while children with high cognitive ability may be able to apply different strategies to learn vocabulary, they may still struggle with reading and spelling. The question then arises, what about those children whose cognitive ability is impaired or those who are not as adept at trying different strategies, as appears to be the case with children with FASD? More recently it has been argued that the phonological loop plays a key role not only in language comprehension, but also in initial language development (Baddeley, Gathercole, & Papagno, 1998). While language learning is not the focus of this research, it is relevant if deficits in the process of language acquisition can be attributed to memory deficits.

The Visual-Spatial Sketchpad. The visual-spatial sketchpad has also been the focus of current research on memory. This system is believed to have two distinct components, object and spatial memory (Baddeley, 2002). The case for a specialized visuospatial component has benefited from the use of a task originally described by Brooks (1967). Named the Brooks Matrix Task, it involves asking participants to imagine a square matrix pattern, and then imagine placing consecutive numbers in adjacent squares. This image may then be used as an aid to recall sentences. To illustrate, the first three sentences may be: "In the starting square put an A"; "In the next square to the right put a B"; "In the next square down put a C." Interference was introduced by having the words, 'up, down, left, and right' replaced by the words 'good, bad, slow, and quick' thus creating nonsense sentences. Recall of these sentences is therefore not aided by the use of the matrix image. This task is considered by many to be a definitive task for the visuospatial scratchpad particularly as it pertains to spatial information (Baddeley & Lieberman, 1980; Logie, Zucco, & Baddeley, 1990; Quinn, 1994; Quinn & Ralston, 1986). This study illustrates some of the empirical support for the visuospatial aspect of Baddeley and Hitch's working memory. Physiological evidence also supports the existence of a specialized visual processing system in which spatial and object memories are subject to selective interference (Tresh, Sinnamon, & Seamon, 1993). Baddeley (1999) has also suggested that mental imagery may play a role in our long-term memory. In this way, learning may be affected at both stages of memory—working and long-term—and therefore may also have cognitive and behavioural consequences.

The Central Executive. The central executive, as noted earlier, is believed to regulate the phonological loop and the visuospatial sketchpad and relates them to long-term memory. Because of the more fluid nature of this system it has reportedly been more difficult to study and is therefore less well understood. One approach has been to look at the combination of memory and processing. To do this studies have focused on memory capacity, believing that differences in capacity relate to differences in performing complex tasks such as comprehension or problem solving (Baddeley, 1999). A more recent attempt to understand the central executive system, has focused on the attentional control characteristics, in part with help from a pre-existing model of attention proposed by Norman and Shallice (Baddeley, 2002). As such, Baddeley identifies three attentional functions of the central executive: (1) the capacity to focus available attention (e.g. Baddeley, Emslie, Kolodny, & Duncan, 1998); (2) divided attention (e.g. Baddeley, Baddeley, Bucks, Wilcock, 2001; Perry & Hodes, 1999); and (3) switching attention. The role of switching attention, however, has not yet been clearly established as a central executive function. In fact, the phonological loop has been identified as possibly playing an important role in controlling action. Consequently, it is currently unknown whether task switching is an executive process or a range of processes (Baddelely, 2002).

Other recent research has revealed the possible existence of a fourth role for the central executive (Baddeley, 2000; 2002). This theoretical fourth component, named the episodic buffer, is believed to form an interface between the visuospatial and phonological subsystems and long-term memory. In doing so, the episodic buffer is assumed to be capable of combining information from long-term memory with that from the slave systems, although future research is necessary to better understand this proposed component (Baddeley, 2002).

Long-Term Memory

Although working memory is the focus of the present study, one other aspect of memory must be elaborated on briefly, that of long-term memory. While working memory is more a process of mental manipulation, encoding, and short-term storage of information, long-term memory involves processes of storage, consolidation, and retrieval (Lezak, 1995). This aspect of memory is often evaluated through the use of delay in memory tasks.

In reviewing the literature on memory research with children with FAS or ARND, the processes of working memory will be referred to specifically where applicable. Long-term memory will be considered only as that construct identified through tasks requiring delay and in terms of types of errors noted in delayed recall. The specific processes of long-term memory will not be considered as they are beyond the scope of this research.

FASD and Memory

While a definite pattern of memory impairment has not yet been established for children with FASD, researchers have begun to explore some of the deficits present. Mattson et al. (1996), using the California Verbal Learning Test (CVLT-C), found that children (5-16 years) with Fetal Alcohol Syndrome (FAS) had difficulty learning and recalling words after a delay, showed increased numbers of intrusion and perseverative errors, and identified more false positives during the recognition portion of the test as compared to the control group. As well, they found these deficits persisted even when intelligence was considered. In other words, intelligence (as measured by IQ) was ruled out as the reason for the memory deficits. In addition, they noted that while the children had difficulty learning the information, once in their memory they seemed able to recall it after a twenty-minute delay. As such, the researchers noted that it appeared as though the deficits were in the process of encoding verbal material during learning, rather than with information recall. This is consistent with earlier findings suggesting that deficits in learning and memory lie at the encoding and storage level (Mattson et al., 1992).

Recent research by Mattson and Roebuck (2002) using the California Verbal Learning Test, the Biber Figure Learning Test, and the Wide Range Assessment of Memory and Learning, has produced several conclusions consistent with earlier findings. They noted that while children with FASD learned less then children without problems, and reached a learning plateau at an earlier level, they nonetheless continued to acquire more information at each additional learning trial. From this they concluded that children with FASD do benefit from multiple opportunities to learn information. They also stated that while the FASD group learned less information, they retained as much verbal information as the controls over the delay period. This again suggests that the deficit is with the process of learning, or encoding, information within short-term memory rather than the long-term storage of information, and is consistent with other studies which have examined verbal memory in FASD (Kaemingk, Mulvaney, Halverson, 2003).

In terms of visual memory, Uecker and Nadel (1996) administered the Memory for 16 Objects, a task that requires the child to recall the name of the object, as well as its placement among others, and other visuospatial measures. From these measures they determined there were immediate but not delayed object recall deficits, general spatial memory deficits, and a significantly distorted spatial array. Interestingly, no deficits were identified for facial recognition tasks.

In addition to spatial deficits, these results seem to also indicate encoding or learning deficits as described in research focusing on auditory verbal memory. In later research, Uecker and Nadel (1998) concluded that, as in animal models, children with FAS demonstrated a spatial but not object memory impairment. The possible impairment of immediate object recall, as well as spatial recall problems, may implicate the visuospatial sketchpad. In his research using rats that had been exposed to alcohol in utero, Nagahara and Handa (1997) identified delay-dependent memory deficits on spatial tasks, although he added that these deficits may decrease as the rat ages.

Using a battery composed of measures of attention and memory, Streissguth et al. (1994) found that fluctuating attentional states, problems with response inhibition, and spatial learning showed the strongest association with prenatal alcohol exposure. Each of these areas plays an important role in the functioning of working memory. Authors of recent research have stated that there are consistent deficits in working memory in children diagnosed with FAS (e.g., Jacobson & Jacobson, 1999). This preliminary research indicates that not only are memory deficits present in these children, but that not all areas of memory are affected. However, as yet there has not been clarity as to the definitions being used to define working memory since much of the research in this area with FASD is not theory-driven. Instead the research originates in the more medical or neurological models, thus the focus is on the neurological structural evidence of damage. While important, this neglects some of the valuable empirical information that does more to explain the functional implications of memory systems.

Kodituawakku, Handmaker, Cutler, Weathersby, and Handmaker (1995) arrived at their conclusions about memory deficits through a study in which they were seeking a better understanding of attentional deficits in FASD. Working with a research model by Norman and Shallice that posits two attentional systems, Kodituwakku et al. stressed the role of attention in behaviour. They focus on the supervisory attentional system, which is fundamental to deliberate conscious control, specifically planning, decision-making, and troubleshooting. The researchers compared ten children having FAS/Fetal Alcohol Effects (FAE) with ten control children. The participants with FAS/FAE were relatively high functioning and did not significantly differ from controls with respect to receptive vocabulary. One goal of this study was to advance understanding of attentional deficits among persons with FAS/FAE. Surprisingly, the children did not show impaired performances on all tasks that were especially sensitive to self-regulatory abilities, a key element of attention. Instead, the children made more perseverative errors on the Wisconsin Card Sort Test, suggesting a difficulty with shifting response sets, and also had difficulty with tasks that evaluated planning ability and the manipulation of information in working memory. They performed equally well on the delayed-response tasks that require sustained attention and the subject-ordered test. Based on these results, Kodituawakku et al. suggest that the FAS/FAE group was best distinguished from the control group by that mechanism which enables us to manage goals in working memory in a flexible manner. The researchers refer to the previously described working memory model proposed by Baddeley and Hitch, and compare the central executive system to the supervisory attentional system. In essence they suggest these two theoretical systems share similar roles, and it is at this point that memory theory and attention theory appear to overlap. In short, Kodituawakku et al. proposed that there exists a dysfunction in the ability of children with FASD to hold and manipulate information and to manage goals in working memory as the underlying cognitive mechanism responsible for the impairments they observed—including those traditionally attributed to attentional deficits. While exciting, these conclusions remain limited in their application due a small sample size which restricts the ability to form generalisations from this research. As such, they encourage further research into these areas.

Several researchers have identified many possible areas of specific impairment within the memory systems of children who are alcohol effected (e.g., Uecker & Nadel, 1998). Unfortunately, given the small sample sizes typically used, the variations in severity, as well as the lack of theoretical underpinnings, it is difficult to decide, based on existing research, whether the deficits identified form a pattern of impairment, and whether this pattern is consistent with the current theories regarding the organization of memory. Furthermore, are there, as Kodituawakku et al. suggest, specific memory processes that can be better understood when viewed in the context of memory theory?

The present study is the first to address key issues to understanding memory deficits in children with FASD by assessing memory within an empirically accepted theoretical framework and by using the Children's Memory Scale (CMS). The CMS is a comprehensive memory measure that utilizes the Baddeley and Hitch model of working memory as its foundation and has thus operationalized this theory of memory. Several aspects of memory can be examined and then interpreted within the Baddeley and Hitch model, and in doing so provide theoretical as well as clinical information regarding the presence or absence of patterns of memory deficits in FASD that will be valuable to researchers and diagnosticians. Specifically, three areas of memory will be examined to identify features within each, as well as their relationship to each other and to their overall memory performance. These three areas include: (1) visual memory; (2) verbal memory; and (3) learning or encoding of information. In addition, these three areas of memory will be studied in a group of children diagnosed using a best practices approach to diagnosis (Astley & Clarren, 1997), thus ensuring accurate identification as well as allowing for clear comparison in future research.

The Present Study

First, two components of visual memory will be examined. While not always consistent, researchers have suggested in preliminary studies that spatial memory is more impaired than visual memory in children with prenatal alcohol exposure (e.g., Uecker & Nadel, 1998). This implicates the visuospatial sketchpad, in which Tresh, Sinnamon, and Seamon (1993) determined that spatial and object memories are, at least in part, distinct systems. This indicates that in children with FASD while the subsystem for spatial information may be impaired, the subsystem for visual information may be preserved. To examine this, two components of the visual memory index will be analyzed using the CMS: dot locations and faces. It is expected that scores on the dot locations subtest, which measures memory for spatial localization, will be more impaired than scores on the faces subtest which measures facial recognition (object memory), for children with FASD.

Second, two components of verbal memory will be examined. Two subtests within the verbal memory index will be compared: stories and word pairs. Word pairs is most directly linked to the articulatory rehearsal process in the phonological loop and is one of the easier tasks on the CMS as clients are prompted with the first word in the pair. Stories on the other hand involves more complex recall as comprehension also plays a role. Thus, the phonological working memory is implicated. This latter subtest will likely be more difficult for alcohol affected children, and perseverative and intrusion errors are likely to be numerous as they have frequently been identified in other studies with this population (e.g. Streissguth et al., 1994). These types of errors are consistent with a failure or inability to shift attention or inhibit responses, functions performed by the central executive component of working memory which is responsible for the attentional functions of memory (e.g., Baddeley, Baddeley, Bucks, & Wilcock, 2001). It is expected that scores for the stories subtest will be significantly lower than those for word pairs. This difference will persist into the delayed components of these subtests.

Finally, learning or encoding will be examined. Some research has suggested that memory deficits in children with FASD lie at the encoding level (e.g., Mattson et al., 1992; Mattson & Roebuck, 2002). This will be examined by comparing immediate index scores to delayed index scores (measures repeated after thirty minutes) for both visual and verbal areas, and by comparing the learning index to the general memory score. As such, immediate memory will be more indicative of their working memory and their ability to acquire the information using the visuospatial sketchpad and the phonological loop, while delayed memory will rely on retrieval abilities as they will have been unable to continue rehearsing the information to keep it in working memory (Cohen, 1997). It is expected that immediate scores will be more impaired than delayed scores, and that this difference will be more pronounced for verbal memory. In all areas of this study, gender will be evaluated since it has been reported as a factor in the normative data used in assessment of memory functioning (Lezak, 1995). Because the CMS does not differentiate between genders, this analysis will rule out the possible influence of this variable. It is expected that gender will not play a significant role in the children's performance on this measure as it is likely that individual differences, as well as the effects of prenatal exposure to alcohol, will be greater than the influence of gender.

In short, three types of memory processes—verbal, visual, and learning—will be examined in this study in an effort to better understand the unique nature of memory in children with FASD. In addition, the children in the study will be identified using the most current diagnostic system to ensure clarity and consistency within the sample. Thus, this research will emphasize the differences between memory functions within this unique population, which will illuminate the need to be comprehensive when assessing memory functions in children with FASD.

Methods

Participants

Thirty children with FASD participated in the study. The participants with FASD were recruited through a private practice that specializes in FASD diagnosis in conjunction with a community-based FASD diagnostic team. As a unique and important component of the current study the participants were diagnosed using criteria based on the 4-Digit Diagnostic Code (Astley & Clarren, 1997). All participants were born to mothers who abused alcohol during pregnancy. This was determined through maternal reports, medical records, or information provided through Child Welfare. In the selection of participants the following criteria were used: fluency in English, no significant hearing or visual impairments, a minimum IQ score of 70 (Borderline range or above) as measured on the Wechsler Intelligence Scale for Children (WISC-III), and no current diagnoses of severe psychiatric disorders (e.g., psychosis). In order to meet these criteria, a significant amount of screening was required, and consequently it required fourteen months to compile the current sample of 30 children from 64 children who were screened. Thirty children tested were found to have IQ scores below 70 and consequently were not included in this study. In addition 4 children withdrew their assent or were not compliant with testing procedures and were therefore not included in the study. The participants had a mean age of 12 years 10 months, and ranged in age from 9-0 to 16-11. There were 14 females and 16 males. Their scores on the WISC – III ranged from 71 to 107, with a mean standard score of 84.13.

Measures

Four Digit Diagnostic Code. All children in this research were diagnosed using the standardized criteria developed and recommended by researchers at the University of Washington (Astley & Clarren, 1997). Briefly, this system documents the magnitude of expression of the four key components of the syndrome, specifically: (1) growth impairment, (2) the FAS facial phenotype, (3) evidence of brain damage, and (4) prenatal alcohol exposure, on separate four-point Likert Scales (Astley & Clarren, 2000). A rank of "1" on any scale means a finding within the normal range. A "4" on any scale indicates a finding that corresponds with accepted cases of FAS. A score of "2" or "3" specifically defines intermediate steps between typical and atypical presentation of FAS characteristics. These scales do not necessarily measure increasing severity, rather they are scales of greater confidence that FAS is present, and as such, a diagnosis of FAS requires ranks of 3 or 4 in all categories. There are, however, many other possible alcohol-related diagnoses provided depending on the code obtained, in which case, there is much more room for consideration of the entire spectrum of this disorder.

All of the children in this study received a brain score of '3' indicating the presence of probable brain dysfunction based on performance of greater than two standard deviations below the norm in intelligence, achievement, adaptation, neuropsychological measures, and/or language. Also required were alcohol exposure scores of '3' indicating that while alcohol use during pregnancy is confirmed the exact amounts consumed are unknown. Together, these two scores are sufficient to indicate the presence of Alcohol Related Neurodevelopmental Disorder (ARND) in all children included in this study. Scores on growth and facial features were not considered as criteria for the purposes of this study, although no children had scores of '4' on either scale indicating that no children in this study presented with 'full FAS.'

*Assessment of Memory. Children's Memory Scale (*CMS; Cohen, 1997*).* To assess memory, the Children's Memory Scale was used. The Children's Memory Scale is a nationally standardized measure normed on 1,000 normally functioning children aged 5 through 16. It is composed of nine subtests that assess functioning in each of four domains: (1) auditory/verbal learning and memory (verbal), (2) visual/nonverbal learning and memory (visual), (3) attention/concentration, and (4) learning. The nine subtests include six core subtests: (1) dot locations, (2) faces, (3) stories, (4) word pairs, (5) numbers, and

(6) sequences. The remaining three subtests are considered supplementary: (7) family pictures, (8) word lists, and (9) picture locations. Each domain is assessed through two core subtests and one supplemental subtest. For each subtest ($M = 10$, $SD = 3$) normative scores are provided for evaluating specific abilities, and eight index scores ($M = 100$, $SD = 15$) are derived from the core subtest scores. In all, the entire measure takes between 45 minutes to one hour to administer.

The six core subtests were administered for this study:

(1) The Dot locations subtest requires the child/adolescent to duplicate a spatial pattern comprised of dots on a grid. Three learning trials are presented, followed by presentation of a distracter grid. The child is then asked to reconstruct the original grid as a measure of immediate memory. After thirty minutes they are again asked to reconstruct the original grid, as a measure of delayed memory. In this way a child's ability to process, learn, and recall spatial locations is assessed.

(2) The Stories subtest entails the child listening to a story, and then retelling it as accurately as they can, which constitutes the immediate memory index. After thirty minutes they are again asked to retell the story to evaluate delayed memory. This subtest assesses the child's ability to process, encode, and recall meaningful, semantically related material.

(3) The faces subtest involves having the child view several pictures of faces which they are asked to remember. They are then shown more pictures at which point they are asked to identify which faces are new and which are faces they recognize. This subtest also involves a thirty-minute delay and retest. This subtest assesses the child's ability to process, encode, and recall human faces.

(4) The word pairs subtest entails having the child listen to a list of word pairs after which they are presented with one of the words from the pair and are asked to recall the word that goes with it. Three learning trials are presented, after which the child is asked to recall as many of the complete work pairs as he or she can. They are asked to do this again after a thirty-minute delay. This subtest assesses the child's ability to process, learn, and recall a list of word pairs.

(5) The numbers subtest requires the child to repeat back a series of number in increasing difficulty. They are then asked to repeat back a series of numbers in reverse order, again in increasing difficulty. This subtest is sensitive to difficulty with attention/concentration and/or auditory/verbal working memory.

(6) The sequences subtest involves having the child perform several types of sequential tasks as quickly as they can without making mistakes. For instance these tasks include listing the days of the week and the months of the year, as well as counting by two's. This task also assesses for difficulty with attention/concentration and auditory/verbal working memory, as well as processing speed.

Reliability coefficients were obtained for each subtest at each age (ranging from 5 - 16 years). The coefficients range from a low of .61 for nine-year olds on that subtest which measures learning of dot locations, to a high of .94 for 13 and 14 year olds on that subtest which measures learning word pairs. Reliabilities were also computed for the average across the ten age-groupings. In this case the range was from .91 for learning word pairs to .71 for the immediate recall of faces. Reliability coefficients were also computed for the eight index scores, for both individual ages, as well as across age-groupings. Reliability of the index scores, as averaged for the across age-groupings ranged from .76 for both visual immediate and visual delayed memory, to .91 for general memory. An interscorer reliability study was also designed and implemented and the data provided shows that the interrater consistency is very high (Cohen, 1997).

CMS scores were also assessed for their stability over time (Cohen, 1997). Drawing from three age bands, 5-8 years, 9-12 years, and 13-16 years, 125 children were retested. The mean test/retest interval was 59.6 days. According to the data provided, CMS indexes generally possess adequate stability across time and age-groups. Two areas however, visual immediate and delayed indexes, were reportedly lower than would be expected. Cohen suggests that this could be due in part to the nature of memory testing (e.g., procedural learning effects and re-exposure to learned material) and psychometric considerations.

Construct validity was assessed through subtest and index intercorrelations (Cohen, 1997). Results of the correlation analysis indicate moderate to high positive relationships between immediate and delayed recall measures within each subtest. The General Memory Index exhibits a moderate to high correlation with other memory indexes (ranging from .68 to .79). Correlations are higher within domains than between domains for all indexes.

Assessment of Cognitive Functioning. Weschler Intelligence Scale for Children – Third Edition (WISC-III; Weschler, 1991*)*. The WISC-III is used to evaluate cognitive functioning through the administration of as many as thirteen subtest. All core subtests were administered in order to obtain a Full Scale score, which was used as a part of the process for screening. These subtests make up a Verbal Scale and a Performance Scale as two important dimensions of cognitive ability. The Verbal Scale includes five subtests: Information, Similarities, Arithmetic, Vocabulary, and Comprehension. The Performance Scale also includes five subtests: Picture Completion, Coding, Picture Arrangement, Block Design, and Object Assembly. Three supplementary subtests are also included, Digit Span, Symbol Search, and Mazes. The WISC-III was standardized on 2,200 children, using sampling methodology that has been described as excellent (Sattler, 2001). In addition, the WISC-III is described as having outstanding reliability, with internal consistency reliability coefficients of .89 and above, and good test-retest correlations being reported, indicating both short- and long-term stability of WISC-III scores (Sattler; Weschler). Good construct validity is also described (Sattler; Weschler).

Procedure

Children and adolescents were tested as a part of a neuropsychological assessment. The measures were administered either by the author or another trained examiner. Both the CMS and the WISC-III were administered using the standardized instructions, and were administered in the same order for every child/adolescent. Testing lasted between two and three hours for each child/adolescent. All testing procedures were approved by the Research Ethics Board at the University of Alberta, and informed consent and assent were obtained from parents and children, respectively, before beginning their involvement in this study (See Appendix A). All guardians and children were informed of their right to withdraw from the study at any time if they so chose.

Results

Within the context of a theoretical model of working memory, three questions were examined in the present research, with a sample of thirty children with FASD: (1) is there a difference between spatial and object memory? (2) is there a difference between memory for simple verbal material and verbal material requiring comprehension? and (3) can the memory deficits observed be attributed to problems with learning rather than long-term memory systems? Analyses were conducted separately for each question.

Visual Memory

To examine the first question regarding spatial and object/facial recognition memory, a 2(Gender) x 2(Task) x 2(Time) ANOVA was used with repeated measures on the last two factors. The between subject factor was gender while the within subject factors were time and task. Thus visual memory was examined through comparison of standard scores (*M* =10, *SD*=3) on both the immediate and delayed components of the dot locations and faces subtests of the CMS to determine whether any difference could be identified between the children's performance on these two subtests. Table 3-1 depicts the descriptive statistics for these subtests.

Table 3-1

Immediate and Delayed Visual Memory for Males and Females by Subtest

		Immediate		Delayed	
Subtest	*n*	*M*	*SD*	*M*	*SD*
Dot locations Male	16	8.25	3.01	8.81	2.95
Female	14	8.21	3.07	7.93	3.69
Total	30	8.23	3.03	8.40	3.29
Faces Male	16	9.44	2.92	9.56	2.48
Female	14	8.79	2.61	8.50	4.09
Total	30	9.13	2.75	9.07	3.31

It was predicted that standard scores on the dot locations subtest (dots) that are measuring spatial memory would be significantly lower than those for the faces subtests (faces) which are measuring object memory, across both time trials. Results from this analysis are illustrated on Table 3-2.

Table 3-2

Analysis of Variance for Visual Memory

Source	*SS*	*df*	*MS*	*Power*	*F*	*p*
			Between Subjects			
Gender	12.95	1	12.95	.14	.783	.38
error	463.09	28	16.54			
			Within Subjects			
Task	17.71	1	17.71	.20	1.35	.26
Task x Gender	1.18	1	1.18	.06	.09	.77
Error (task)	366.66	28	13.10			
Time	2.52	1	2.52	.05	.005	.94
Time x Gender	2.96	1	2.96	.12	.64	.43
Error (time)	129.22	28	4.62			
Task x Time	.36	1	.36	.06	.072	.79
Time x Task x Gender	.36	1	.36	.06	.072	.79
Error (task x time)	139.48	28	4.98			

While the means for dots were slightly lower than faces no main effect was found for the within subject factors of task [($F(1,28) = 1.35$, p = ns)] and time [($F(1,28) = .01$, p = ns)]. Furthermore, no main effect for the between subject factor, gender, was identified [($F(1,28) = .78$, p = ns)]. Similarly, no interaction effects were found for task and gender [($F(1,28) = .90$, p = ns)], for time and gender [($F(1,28) = .64$, p = ns)], for task and time [($F(1,28) = .07$, p = ns)], or for task, time, and gender [($F(1,28) = 1.35$, p = ns)]indicating a similar pattern of visual memory functioning for both types of visual memory, at both immediate and delayed times.

Verbal Memory

The second question, evaluating verbal memory, was also examined using a 2(Gender) x 2(Task) x 2(Time) ANOVA design with repeated measures on the last two factors. Again, the between subject factor was gender while the within subject factors were time and task. In this way, verbal memory was examined through comparison of immediate and delayed standard scores on the stories and word pairs subtests of the CMS. Descriptive statistics for these subtests are presented in Table 3-3.

Table 3-3

Immediate and Delayed Verbal Memory for Males and Females by Subtest

		Immediate		Delayed	
Subtest	*n*	*M*	*SD*	*M*	*SD*
Stories Male	16	9.50	3.46	9.19	3.15
Female	14	8.50	2.25	8.86	2.28
Total	30	9.03	2.95	9.03	2.74
Word Pairs Male	16	6.06	3.13	6.81	3.04
Female	14	6.64	2.90	7.93	3.05
Total	30	6.33	2.99	7.33	3.04

It was expected that standard scores on both the immediate and delayed stories subtest (stories) would be significantly lower than those for the word pairs subtest (words), which would indicate that the children performed better on the simple verbal task than on the verbal task requiring comprehension. Results from this analysis are described in Table 3-4.

Table 3-4

Analysis of Variance for Verbal Memory

Source	*SS*	*df*	*MS*	*Power*	*F*	*p*
			Between Subjects			
Gender	.25	1	.25	.05	.015	.90
error	459.72	28	16.42			
			Within Subjects			
Task	138.00	1	138.00	.80	8.47	< .01
Task x Gender	17.10	1	17.10	.17	1.05	.31
Error (task)	456.20	28	16.29			
Time	8.08	1	8.08	.84	9.31	< .01
Time x Gender	2.71	1	2.71	.40	3.13	.09
Error (time)	24.29	28	.87			
Task x Time	7.4	1	7.4	.63	5.61	.03
Time x Task x Gender	3.35	1	3.35	.05	.03	.88
Error (task x time)	36.97	28	1.32			

In this analysis a main effect was found for Task [($F(1,28) = 8.47$, $p < .01$)] and Time [($F(1,28) = 9.31$, $p < .01$)] indicating that the children did better at time 2 (delayed) than time 1 (immediate), and that the children did better on the stories subtest than the word pairs subtest. No interaction effects were found for task and gender [($F(1,28) = 1.05$, p = ns)], for time and gender [($F(1,28) = 3.127$, p = ns)], or for task, time, and gender [($F(1,28) = .025$, p = ns)]. However, an interaction effect was identified for task and time [($F(1,28) = 5.605$, $p < .05$)] indicating that performance between the different times (immediate and delayed) was not the same for both tasks. Specifically, no difference was identified for stories as the scores were identical ($M = 9.03$ for both), while a difference was observed for words ($M = 6.33$ for immediate and $M = 7.33$ for delayed) as can be seen in Figure 3-2.

Figure3-2. Verbal Memory Tasks at Two Intervals.

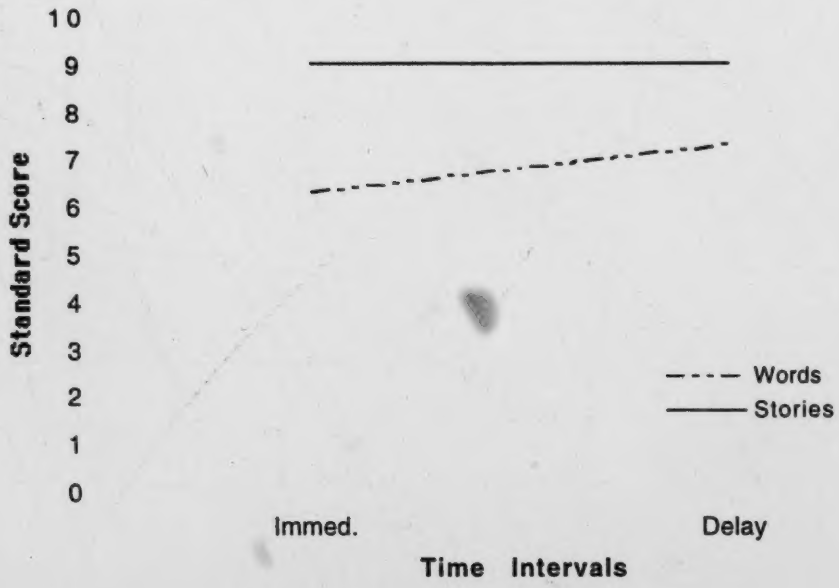


Figure 3-2. Children with FASD performed significantly better on a task of story recall than on a task of word recall. On the task of word recall, the children performed significantly better at time 2 (delay) than time 1 (immediate), while their performance on the stories task was identical for both times.

No main effect was identified for gender, the between subjects variable [(*F*(1,28) = .015, *p* = ns)]. Therefore, a 2 (Task) x 2 (Time) ANOVA design with repeated measures was then conducted for stories and word pairs. A main effect was again identified for task [(F(1,29) = 8.90, p <.01)] and time [(F(1,29) = 8.06, p <.01)], and an interaction effect was identified for task and time [(F(1,29) = 5.88, p < .05)]. These results are presented in Table 3-5. To better understand the interaction effect observed dependent t-tests were conducted at time one and time two. This analysis revealed that there was a significant difference between stories immediate and words immediate ($p < .01$), but that there was not a significant difference between stories delayed and words delayed ($p = .03$). This indicates that the children's performance for immediate words was significantly lower than their performance on any other verbal task.

Table 3-5

Analysis of Variance for Stories and Word Pairs

Source	*SS*	*df*	*MS*	*Power*	*F*	*p*
			Within Subjects			
Task	145.20	1	145.20	.82	8.90	< .01
Error (task)	473.30	29	16.32			
Time	7.50	1	7.50	.78	8.06	< .01
Error (time)	27.00	29		.931		
Task x Time	7.50	1	7.50	.65	5.88	< .05
Error (task*time)	37.00	29	1.28			

Learning

The third question, regarding learning, was analysed in two ways. In the first analysis, a 2(Task) x 2(Gender) ANOVA design was used. The between subject factor was gender and the within subject factor was task. As such, indexes of general memory and general learning from the CMS were compared. The descriptive information is presented in Table 3-6.

Table 3-6

Learning for Males and Females by Index

Subtest	*n*	*M*	*SD*
General Memory Male	16	86.75	11.65
Female	14	85.14	13.03
Total	30	86.00	12.13
Learning Male	16	83.31	13.45
Female	14	83.29	10.72
Total	30	83.30	12.04

It was expected that if the greatest problems for the children with FASD lay in the acquisition of information then their scores on the learning index would be significantly lower than their scores on the general memory index. On the CMS, the learning index is generated through a measure of the amount of information learned during learning trials. It does not incorporate scores of immediate memory or delayed memory and is therefore independent of the general memory score that relies on these factors. No main effect was found for task [($F(1,28) = 1.588$, $p =$ ns)] and no interaction effect was found between task and gender [($F(1,28) = .142$, $p =$ ns)]. This indicates that no significant differences were identified between the children's performance in these two areas. In addition, no between subject main effect was found for gender [($F(1,28) = .04$, $p =$ ns)]. These results are presented in Table 3-7.

Table 3-7

Analysis of Variance for Learning

Source	*SS*	*df*	*MS*	*Power*	*F*	*p*
			Between Subjects			
Gender	9.97	1	9.97	.06	.04	.84
error	6606.18	28	235.94			
			Within Subjects			
Task	104.66	1	104.66	.23	1.59	.22
Task x Gender	9.32	1	9.32	.07	.14	.71
Error (task)	1844.83	28	65.89			

In the second analysis of learning, a 2(Gender) x 2(Task) x 2(Time) ANOVA design was used with repeated measures on the last two factors to determine whether there was any significant difference between working memory (visual/verbal immediate) and long term memory (visual/verbal delayed) as measured on the CMS. This was intended to further understand whether there is a difference between the children with FASD's ability to learn information (immediate) and their ability to recall and retrieve information after a delay (delay) on verbal and visual tasks. Again, the between subject factor was gender, while the within subject factors were time and task. Thus, delayed and immediate, verbal and visual indexes were compared. Descriptive information is provided in Table 3-8.

Table 3-8

Visual and Verbal Learning for Males and Females by Index

		Immediate		Delayed	
Subtest	*n*	*M*	*SD*	*M*	*SD*
Visual Male	16	92.88	14.16	95.38	12.24
Female	14	90.86	10.22	88.93	18.13
Total	30	91.93	12.49	92.37	15.34
Verbal Male	16	86.50	13.42	87.88	11.79
Female	14	85.21	12.18	92.21	12.26
Total	30	85.90	12.65	89.90	12.00

No main effects were identified for time [($F(1,28) = 2.847$, p = ns)], task [($F(1,28) = 1.587$, p = ns)], or gender [(F(1,28) = .19, p = ns)]. In addition, no interaction effects were identified for task and gender [($F(1,28) = .799$, p = ns)], for time and gender [($F(1,28) = .05$, p = ns)], for task and time [($F(1,28) = 1.99$, p = ns)], or for task, time, and gender [($F(1,28)=3.31$, p = ns)]. Again, this indicates that the children's performance was similar for both task and time. These results are presented in Table 3-9.

Table 3-9

Analysis of Variance for Visual and Verbal Learning

Source	*SS*	*df*	*MS*	*Power*	*F*	*p*
			Between Subjects			
Gender	54.65	1	54.65	.07	.19	.66
error	7935.53	28	283.41			
			Within Subjects			
Task	491.83	1	491.83	.23	1.59	.22
Task x Gender	247.63	1	247.63	.14	.80	.38
Error (task)	8675.74	28	309.85			
Time	149.41	1	149.41	.37	2.85	.10
Time x Gender	2.67	1	2.67	.06	.05	.82
Error (time)	1469.17	28	52.47			
Task x Time	113.67	1	113.67	.28	1.99	.17
Time x Task x Gender	188.67	1	188.67	.42	3.31	.08
Error (task x time)	1596.17	28	57.01			

Discussion

Research on memory in children with FASD has produced varying results in an effort to identify some pattern of impairment. While there has been disagreement, some consistent themes are beginning to emerge, specifically theories that memory deficits may be found primarily in the process of encoding or manipulating information in short-term memory, rather than with long-term storage of information (e.g., Mattson & Roebuck, 2002). In the present paper it was intended to further explore memory functions and expose possible patterns that may exist in children with FASD. As such, a comprehensive memory test, the Children's Memory Scale, was used to explore visual and verbal memory, as well as learning and encoding, with an FASD population. In addition, a model of working memory (Baddeley & Hitch, 1974) was applied. This theoretical framework is a valuable means through which hypotheses can be tested and interpreted in a consistent manner.

Visual Memory

Examination of visual memory revealed no significant differences between the types of visual memory (spatial and object) or between different times (immediate and delayed) examined. This suggests there is no consistent pattern in the types of memory deficits present in children with FASD, in contrast to results found by Uecker and Nadel (1996, 1998). In their study, Uecker and Nadel described a general spatial memory deficit evident in difficulties reproducing a spatial array and no difficulties with facial recognition for children with FASD when compared to a control group. In addition, they reported differences between immediate and delayed visual memory in children with FASD, which could be indicative of encoding deficits. While the present research finds no support for the distinction in deficits between types of visual memory, it does not necessarily indicate the absence of encoding deficits, as evaluated through comparison between immediate and delayed memory. Even though delayed memory was not found to be less impaired than immediate memory, it was also not found to be more impaired. As such, there was no indication that the retrieval of information that was committed to long-term memory contributed to the deficits observed. Furthermore, observation revealed that the children tended to recall the same amount of information after the delay as before, suggesting that they did not lose a lot of information during the delay. Therefore it is plausible that the problems are in information acquisition, or the encoding process. This conclusion is consistent with other research in this area (e.g. Mattson & Roebuck,

2002) which found more indication of memory deficits in the process of learning rather than recalling information in children with FASD.

Further complicating this issue of encoding deficits in visual memory processes is research by Mattson and Roebuck (2002). Mattson and Roebuck identified deficits for delayed recall, beyond what could be explained by acquisition deficits, suggesting some decay of visual information. This difference, however, may reflect the dissimilarity in the measures administered. A significant difference is the drawing requirement in the Mattson study, which was not a component of the current study. In addition, the Mattson study involved memory for geometric figures, while the current study involved a spatial localization task and facial recognition.

Two issues emerge from the variation in results observed in the research into the visual memory system. First, there is a wide range of visual memory tasks available, all of which are described as visual memory. Consequently, with the possibility of such diversity within this domain, specific description of the types of deficits will be necessary to obtain an accurate picture for both clinical and research purposes. Second, from the perspective of the working memory model by Baddelely and Hitch, it is difficult to know how best to separate these components within the visuospatial sketchpad. For instance, the dot locations task appears consistent with the spatial component of the sketchpad. However, it has been noted that storage of a spatial pattern and finding a path through a pattern may be different functions (Smyth & Pelky, 1992), suggesting this test alone does not provide a full picture of spatial memory. Facial recognition is even more of an enigma. The organization of facial features could encompass elements of spatial memory skills, whole object memory, or both. Likely, both spatial and object aspects of memory are implicated, in which case this task illustrates the interaction that occurs between the components of visual memory. A plausible explanation may be that deficits in one specific system are, to some degree, compensated for by another. In turn, this may result in the appearance of inconsistent behaviour in children with FASD, which may be the result of subtle situational or contextual differences that affect the ability of different systems to compensate for one another. This may also explain why other research with children with FASD has found differences between spatial recall and facial recognition that were not apparent in the current study (e.g., Uecker & Nadel, 1996). Nonetheless, difficulty in defining the specific components of visual memory and their interconnections has also been described within the working memory literature (e.g. Baddelely, 2002), confirming the functional complexity of our visual memory system on a theoretical level, in addition to the clinical level observed here.

At this point it should be noted that while differences between the different components in visual memory were not identified, this does not suggest the lack of deficits in visual memory in children with FASD. In fact, looking at standard scores for this population, the average was lower that the standardized mean in all areas; however the lack of a control group prevents any conclusions in this regard. In addition, the analyses conducted in this study were found to have little statistical power, suggesting that it is possible that an existing relationship such as the ones described here may exist but was not identified. This may have been due to small sample size, low instrument sensitivity, or high sample variability.

Verbal Memory

In contrast to the visual memory components studied, there was a significant difference between types of verbal memory in the FASD sample. Specifically, recall of word pairs was found to be significantly more impaired than that for stories. In addition to this, recall of immediate word pairs was significantly more impaired than that for delayed word pairs. This lends itself to two levels of interpretation. The first is the difference between performance on the two tasks, which was in the opposite direction than predicted. In administering the measures, perseverative errors were noted to be numerous, as expected, however the effects of these errors were different for the two tasks. For the stories, the children tended to 'add in' information that seemed to make sense to them, even when they seemed to be struggling to remember the actual story. In other words, they tended to provide more information than was a part of the story. Consequently, as long as they were able to pick up the general theme behind the story, they were able to generate a similar story line, regardless of whether they truly understood the content. In doing so, it seems as though they were relying less on the phonological loop in their working memory, and more on the newest of Baddeley's proposed functions of the Central Executive, the episodic buffer, which could have allowed them to either combine the new information with previous experience or use chunking strategies. While this may have compromised accuracy it did allow them to recall more information about the story, in addition to a lot of inaccurate, superfluous information for which they were not penalized. This is particularly interesting when we consider that often children with FASD present, in conversation, as more competent than they really are which tends to contribute to unrealistic expectations from those around them (Streissguth, 1997).

Unlike stories, perseverative errors were detrimental in the word pairs task in which accuracy was required. These errors tended to involve using the same word over and over again until they found its match. The children seemed to do better on those items in which the words were related, while they had great difficulty with those pairs in which there was no relationship. Similar to Mattson and Roebuck's (2002) conclusions, it seemed as though in their work the children did continue to acquire information in successive learning trials, rather than reaching a plateau and not acquiring further information after that. Thus, it appears that their greatest difficulty arose with those items in which the phonological loop was required, which would have facilitated learning though internal recitation and adequate phonological storage.

Learning

The second level of interpretation for the verbal subtests is that of immediate and delayed recall. In this case, word pairs were found to have significantly less impairment for the delayed component. Close examination of the scores indicated that this variation was often created when the same amount of information was recalled after the delay as before the delay. Because the immediate memory scores also included an element of learning this is not a pure comparison. Slower learning or acquisition of information would also affect the immediate memory score thus this score tends to reflect the speed with which information is acquired through consecutive trials, rather than just the amount acquired in the end. Consequently, this indicates that it is likely the point of encoding or acquiring verbal information that is the greater problem for children with FASD rather than the process of accurately retaining and retrieving verbal information, a finding consistent with recent research in this area (e.g., Kaemingk, Mulvaney,& Halverson, 2003; Mattson & Roebuck, 2002). Thus, the evidence continues to suggest that the working memory, and in this case the phonological loop, is primarily responsible for deficits observed in children with FASD. Moreover, considering the key role of the phonological loop in language acquisition (Baddeley, Gathercole, & Papagno, 1998), a deficit in this area could be the catalyst for the many language-based problems observed in children with FASD. Further investigation is required to explore this possible relationship.

It is also possible that deficits observed in verbal learning can be attributed to deficits in the Central Executive System. It is the Central Executive System that is posited to regulate our ability to sustain attention, selectively focus, and shift attention. Memory for word pairs requires both sustained and focused attention in addition to the ability to make a subtle shift between word pairs. Difficulty in any of these areas may have contributed to the difficulty observed here, particularly in terms of the tendency of children with FASD to perseverate. Use of this model of memory in future research to more closely study the types of attention deficits in individuals with FASD, from the perspective of a memory model, may help lend some clarity to this disparate area of study.

Further examination of the data, looking at delayed and immediate, verbal and nonverbal memory, as well as learning and general memory, failed to find any additional differences. However, as noted with visual memory, the absence of a difference in these cases does not argue against a difference in these memory systems, but simply that the data from the present study fails to provide any clear evidence in support of these conclusions. Moreover, no significance was found, in any area, for the effects of gender on the results . While not unexpected given the nature of the brain injury present in children with FASD, the consistently low power observed whenever gender was considered indicates that the ability to detect any effects was very low in this study. As such, further research into this area, possibly with a larger sample size, may provide greater insight into this area.

Limitations and Future Directions

A comment must be made about this data in general. Overall, the scores obtained by these children showed a tremendous variability, both between children as well as within individual children, as statistically depicted by the high error scores. This variability clearly makes the quest for a pattern more difficult, and possibly deceptive insofar as it may be inappropriate to apply any pattern to a group that demonstrates such variability. This is not to say that exploration of the specific nature of deficits present is not warranted - it is. It can contribute to our knowledge base and provide insight as we develop effective intervention techniques, though future researchers will need to be aware of the unique features of this population. At the same time, however, caution must be observed, as we must clearly communicate the limits of such a pattern in its application to individual children with FASD. Blind application of any pattern to FASD will only increase the possibility of misdiagnosis and rather than clarifying our conception of FASD will preclude true understanding of this complex disorder.

Memory is fundamental to our daily functioning. It shapes the way we view and interact with the world around us. It defines the way in which we acquire and apply information and skills on a daily basis. In addition, it is complex and interdependent on many different systems in order to be effective. As such, it is an area that warrants continued study and exploration with FASD children. Further research is necessary to continue exploring the deficits that appear to exist at the encoding level of memory, particularly with the phonological loop. In addition, research into language acquisition with FASD children, as it pertains to the phonological loop may provide additional information into the foundation of many learning problems observed as these children negotiate the educational system. Further study using the CMS, or an equally comprehensive memory measure, is encouraged to permit better understanding of the clinical presentation of this population. Use of a control group could also provide valuable information about the ability of this measure to distinguish between FASD and other groups.

In summary, in the data presented in this study it is clear that children and adolescents with FASD displayed specific types of verbal memory deficits. In addition, these deficits were greater for immediate rather than delayed memory. These data are consistent with previous studies that describe deficits in immediate memory, and suggest that deficits in delayed memory are better accounted for by encoding deficits. Furthermore, contrary to previous research, retention of visual information was not fond to be more impaired that verbal. Moreover, spatial acquisition was not fond to be more impaired than facial recognition. Further research into these distinctions in memory is warranted, as is exploration into educational techniques that could account for delayed encoding in children with FASD. In this way practical strategies for children with FASD may be developed and implemented to help these children make the best use of the many abilities they have.

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IV. The Interrelationship of Neuropsychological Deficits in Fetal Alcohol Spectrum Disorders (FASD)

Introduction

Fetal Alcohol Spectrum Disorders (FASD) were first identified in 1973 as a pattern of deficits present in children born to alcoholic mothers (Jones & Smith, 1973). At that time three features were identified as being associated with prenatal alcohol exposure: facial malformation, growth retardation, and central nervous system (CNS) dysfunction. Of these three it is the cognitive and behavioural aspects of the CNS dysfunction that are most relevant to psycho-educational and psychological assessment. These deficits may include disordered motor development, diminished intellectual functioning, delayed and disordered speech and language development, problems in social perception, memory deficits, and deficits in response inhibition and attention (Mattson & Riley, 1998). The Institute of Medicine (1996) specifies that these patterns of behaviour or cognitive abnormalities are complex, inconsistent with developmental level, and cannot be explained by familial background or environment alone.

In the following paper, three areas of CNS dysfunction will be reviewed as they pertain to children with FASD: (1) intelligence, (2) executive functioning, and (3) memory. It is expected that, while there will be great variability between these measures within this population, a relationship between executive functioning and memory will be identified. In particular, it is expected that performance on a memory measure will indicate, to a significant degree, performance on a task of executive functioning. As such, four predictions will be tested in this study: (1) that there will be a positive correlation between measures evaluating memory and executive functioning, (2) that performance on three elements of memory, learning, attention and visual memory will predict performance on a measure of executive functioning, (3) that verbal memory will not predict performance on executive functioning, and (4) that ability as measured using an intelligence test will have no ability to predict performance on either a memory measure or a measure of executive functioning.

In past studies, researchers and clinicians have studied FASD in an effort to identify a consistent pattern of deficits (e.g., Mattson & Riley, 1998). However, for the most part what they have found is just the opposite, a *lack* of consistent deficits. As yet, no single type of CNS damage or pattern of dysfunction has been identified that characterizes all children who have been prenatally effected by alcohol (Streissguth, 1997). This variability could have many origins. Different patterns of maternal alcohol consumption and different levels of individual susceptibility are just two possible factors. Unfortunately, this variability results in unexpected and confusing behaviour in children with FASD when the diagnosis has not been clearly linked to specific structural anomalies in the human brain. Nor have these behaviours been clearly described by current standardized methods of diagnosis, such as the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV). Consequently, many professionals struggle to understand the disorder and its implications.

As researchers continue to bridge the gap between the science and practice of FASD, exploration continues into the functional presentation of deficits in FASD. At the same time investigation is ongoing into the possibility that common underlying mechanisms may be the origin of observed deficits in different areas. Two areas in which some similarities have been identified in the literature are executive functioning and memory. Further investigation into these similarities may provide insight into underlying factors that could be responsible for some of the symptoms observed in both areas, and in so doing contribute to our understanding of FASD. Knowledge of such a relationship, then, can advance our understanding of the fundamental deficits that produce the symptomology observed in FASD. In turn, assessment and then intervention and remediation can be appropriately adapted to identify and respond to the most fundamental deficits in FASD.

In addition to identification of a common underlying mechanism in children with FASD, it is hoped that the comparison of these areas of functioning will emphasize the unusual degree of inconsistency that exists within this population, and consequently, the need to move away from traditional expectations of the relationship between these different areas of functioning and towards a more individualized approach to assessment. Moreover, it is hoped that this study will provide insight into the necessity of using a comprehensive battery of tests in assessing FASD in order to derive a clear picture of the deficits present.

Assessment of FASD

Sattler (2001) states that "assessment is a way of gaining some understanding of the children in order to make informed decisions" (p.3). Similarly, the goal of assessment with children suspected of having FASD is to implement appropriate intervention and prevention programs. Sattler continues by suggesting that diagnosis is one way to do this, and that formal diagnosis tends to depend on a classification system such as is found in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994). Key to this system, Sattler states, is an ordered presentation of group differences that reflect the intrinsic distinctions among individuals within different classifications. It is at this point that the diagnosis of FASD seems to diverge from traditional psychological assessment methods since the sole unifying feature of FASD is etiology rather than any intrinsic distinctions. Unlike most other diagnoses typically made through assessment, the resulting symptomology of FASD will vary along the spectrum to such an extent that it is possible for two children with FASD to share no common symptoms. In addition, this symptomology encompasses both medical and psychological elements, neither of which are clearly defined within standard diagnostic manuals (e.g., DSM-IV).

Consequently diagnosis of FASD is complex and assessment of this population must be comprehensive, with an appreciation for, if not an understanding of, the possible range of underlying brain injury responsible for the deficits observed. For instance the brain injury may be due to structural deficits (e.g., Mattson & Jernigan, 1994; Riley et al., 1995), less dendritic branching, defects in neuronal migration, desynchronization of radial glia into astroglia, and problems with normal myelination in the central nervous system (Archibald, Gamst, & Riley, 2001; Miller, 1986, 1988, 1993). Even neurotransmitter abnormalities have been identified (Xu & Shen, 2001). Clearly the consequences of such a wide array of effects on the brain is likely to be varied, necessitating a thorough assessment of all areas of functioning.

Considering this information, then, it becomes clear as to why traditional assessment techniques have failed to identify a pattern to the deficits present in children with FASD. The effects of alcohol on a developing brain can have many different effects, both direct and indirect. As such the deficits observed may not ascribe to any of our current models of dysfunction, but rather require a new approach to understanding the deficits present. In the case of executive functioning and memory, deficits may be best conceptualized through examination of any mechanisms that influence both areas. If such common mechanisms exist, then they may allow for reframing of our understanding of the roles of executive functioning and memory to achieve a better understanding of the true deficits that are present in FASD. In this way, assessment of FASD may require starting from the brain injury incurred rather than the symptoms observed, followed by the consequences of these deficits to memory and executive functioning, which in turn create the observable symptoms such as cognitive delays, attentional concerns, or behavioural problems. Any resulting intervention would then be able to target the root of the problem. In moving to this level of interpretation, however, greater understanding of the symptoms present in FASD and the way in which they are interrelated is necessary. By better understanding the observable deficits, exploration into possible underlying mechanisms can begin, followed by eventually connecting these fundamental problems with the brain dysfunction at their origin.

Intelligence

Since the mid-1970s, it has become evident that the range of intellectual disabilities associated with Fetal Alcohol Syndrome (FAS), as it was originally named, is very broad, and that no specific level of intellectual functioning could ever reasonably serve as the distinguishing CNS characteristic for defining FAS (Streissguth, Bookstein, Barr, Press, & Sampson, 1998; Striessguth, Herman & Smith, 1978). This broad range of measured intelligence was supported by Mattson and Riley's 1998 review of neurobehavioural deficits in which they examined individual case studies as well as retrospective and prospective studies. They concluded that while intelligence deficits (as estimated by IQ) in children with prenatal alcohol exposure are relatively stable, as yet no clear pattern of intelligence scores (such as performance versus verbal) had been definitively established (Mattson & Riley, 1998). Steinhausen and Spohr (1998) agreed that children with FASD tend to have IQ deficits, and interestingly, they state that "although mental retardation and cognitive deficits are extremely over represented among FAS children, it should not be overlooked that various levels of mental functioning are to be found among this group of children" (p.337). They go on to explain that they have encountered many FAS children who were functioning at age appropriate levels in school. Furthermore, they state that their research does not suggest a linear relation between the degree of morphological damage and intelligence. In other words, although a child may present externally as more severely alcohol affected, he or she may not be more severely impaired in terms of daily functioning and skill levels. Ernhart et al. (1995) followed eight children, identified in infancy by trained clinicians as having FAS, from infancy until they were five years old. They found only one of these eight children was cognitively impaired according to the IQ tests administered. Thus, it seems that researchers have not yet identified a consistent pattern of intellectual deficits in FASD, and that even ~~the~~ extent to which intellectual deficits are present in FASD is disputed. In addition, Kerns and Don (2000) suggest that even if intellectual deficits are identified, these deficits do not provide a complete picture of cognitive functioning, a conclusion they formed after studying sixteen non-retarded young adults with FAS for whom measures of academic and intellectual functioning did not accurately reflect the deficits that were present in the areas of attention, memory, and executive function.

Other research seems to suggest a pattern between the amount of alcohol to which a child is exposed prenatally and the presence and severity of intellectual deficits (e.g. Aronson & Hagberg, 1998). For instance, Aronson and Hagberg, found that the children in their study displayed more frequent and severe disturbance if the mother had abused alcohol throughout pregnancy. These authors suggest that patterns of alcohol consumption may have a direct effect on intellectual impairments. In earlier research Streissguth, Barr, and Sampson (1990) also propose that a relationship exists between the amount of alcohol consumed and the amount of intellectual impairment incurred. They state that "above one ounce of absolute alcohol per day on the average, the magnitude of the IQ decrement…is almost half of a standard deviation (almost seven IQ points)" (p.677). In particular, they state that the Digit Span and Arithmetic subtest scores, as measured on the Weschler Intelligence Scale for Children – Revised (WISC-R), are most highly related to prenatal alcohol exposure. They note that these items have strong memory and attentional components, and arithmetic also involves abstract problem-solving skills. Importantly, they conclude that alcohol-related attentional deficits, observed throughout the child's life, contribute to learning problems as identified in these measures. Thus, it seems that to some degree deficits identified by intelligence tests may not be intelligence deficits at all but rather other processes that depress IQ's. Furthermore, as Sattler (1992) notes, intelligent tests measure only a part of a domain that reflects intelligent behaviour.

Clearly, there are many factors involved in the measure of intelligence, including many environmental and cultural variables that fall outside the purview of the present research. Furthermore, the value of the information obtained through tests of intelligence given to children with FASD is disputed. It seems that at this time there is no indication that intelligence tests tap into those problems that are most debilitating for children with prenatal alcohol exposure. Researchers have begun to conclude that children with FASD can have IQ scores in the average range and still have specific cognitive or neuropsychological impairments or problems with adaptive behaviours that do not register on IQ test scores (e.g., Kaemingk & Paquette, 1999; Streissguth, 1997). Thus, the usefulness of intelligence tests, in isolation, for diagnosis of this population seems limited, in turn making appropriate educational placements more difficult to determine. But when studied alongside a more detailed examination of specific abilities such as executive functioning and memory skills, as is the intention with the present study, the goal is to develop a more global view of functioning as well as inform practitioners of the specific needs that need to be addressed when placement, modification, and remediation are being considered for children with FASD.

Executive Functioning

Executive functioning (EF) includes elements of problem solving and mental flexibility. Deficits in executive functioning may interfere with daily activities in many ways, such as limiting independence, compromising reasoning skills, and hindering effective interaction, regardless of how well preserved the cognitive capabilities are (Lezak, 1995). But impairment in these areas may not always be evident in standard assessment techniques that often focus on specific functions or abilities. Instead, impairments in EF tend to show up globally, affecting all aspects of behaviour. Consequently, EF deficits may be implicated in some of the everyday or adaptive problems identified in FASD, including understanding cause and effect, exercising good judgement, avoiding victimization, being appropriately guarded with strangers, and not acting on impulses (Lockhart, 2001). In some cases EF is also implicated in short-term memory functions. Baddeley and Hitch (1973) propose that a Central Executive System exists to govern attentional systems and integrate short-term memory functions, and consequently plays a significant role within our ability to function on a daily basis. As such, the role of the EF is far-reaching and deficits in this area may play a role in the alcohol-related behavioural problems observed in FASD, such as hyperactivity, impulsivity, attention deficits, and perseveration and intrusive errors that influence both academic and social aspects of an adolescent's life (Mattson, Goodman, Caine, Delis, & Riley, 1999).

The Wisconsin Card Sorting Test (WCST) is often used to measure EF, and requires both problem-solving skills and mental flexibility. When used with an FASD population both adults and adolescents have been observed to be less accurate, achieve fewer categories, and make more perseverative errors (Carmichael Olson et al., 1998; Kodituwakku et al., 1995). However, despite these results, others have found that performance by groups with FASD while somewhat lower than controls was not statistically significantly different (Coles et al., 1997). In addition, their performance level was much higher than expected given their overall level of ability (Mattson, Roebuck, & Riley, 1996)

Additional research using the Delis-Kaplan Executive Function Scale evaluated four areas of EF: planning ability, cognitive flexibility, selective inhibition, and concept formation and reasoning. Not only were deficits identified for children with FASD in all four areas (Mattson et al., 1999), but these deficits could not be explained by difficulties in other areas, such as cognitive impairment. In addition the EF deficits persisted both in the presence and absence of any physical features of FASD. Kodituwakku et al. (1995) also found that children with FASD had difficulty planning ahead and tended to perseverate on incorrect strategies. They went on to suggest that these problems were due to deficits in an underlying mechanism, specifically, that mechanism responsible for holding and manipulating information and managing goals in the working memory in a flexible manner. As such, they implicate the central executive system of the working memory, as proposed by Baddeley and Hitch (1973). As previously noted, the central executive has been described as a governing system within working memory, in which attention is controlled and the necessary systems are integrated. The present study seeks, in part, to connect the deficits in children with FASD to executive functioning, resulting in a better understanding of the way in which this sub-system is affected and the consequences for children with FASD. In turn, this information could be used for the development of remediation strategies that target the source of the deficit(s) rather than only focusing on the symptoms.

Memory

While a definite pattern of memory impairment has not yet been established for children with FASD, researchers have begun to explore some of the deficits present. For example, deficits have been identified for auditory memory (Carmichael Olson, Feldman, Streissguth, Gonzalez, 1992), spatial memory (Uecker, & Nadel, 1996), and working memory (Jacobson & Jacobson, 1999; Kodituwakku, Handmaker, Cutler, Weathersby, Handmaker, 1995). Some of this research indicates that not only are memory deficits present in these children, but that not all areas of memory are affected. For instance, using a battery composed of measures of attention and memory, Streissguth et al. (1994)

found that fluctuating attentional states, problems with response inhibition, and spatial learning showed the strongest association with prenatal alcohol exposure. Here again, there appears to be overlap with the deficits described within EF. Clearly the relationship between EF and working memory is intricate, and deficits in either area may have subtle yet profound effects on a child/adolescent's educational and social functioning.

Using a measure of auditory, verbal memory, Mattson, Riley, Delis, Stern, and Jones (1996) found that children with FAS had difficulty learning and recalling words after a delay, showed increased numbers of intrusion and perseverative errors, and identified more false positives during the recognition portion of the test. In addition, given that these deficits persisted even when mental age was considered, they were able to rule out IQ as the reason for the memory deficits. The researchers noted that it appeared as though the deficits were due to problems with the encoding or learning process, rather than with retrieving or remembering the information, a finding consistent with earlier research (Mattson, et al., 1992).

Further support for this position was provided in more recent research (Mattson & Roebuck, 2002), in which it was observed that children with FASD benefit from repeated exposure to information, and that although the FASD group learned less information, they retained as much verbal information as the controls over the delay period. As such, the researchers again concluded that the observed deficit is with the process of learning, or encoding, information within short-term memory rather than the long-term storage of information.

In terms of visual memory, Uecker and Nadel (1996) have reported immediate, but not delayed, object recall, general spatial memory deficits, and a significantly distorted spatial array in children with FAS. In later research, Uecker and Nadel (1998) concluded that, as in animal models, children with FAS demonstrated a spatial but not object memory impairment. This is consistent with earlier research by Streissguth et al. (1994) and indicates that not all areas of memory seem to be similarly affected by prenatal alcohol exposure.

In short, while no clear patterns of memory deficits have been described for FASD, researchers have begun to suggest that the impairment is not with long-term memory but rather with the process of information acquisition. As such, similar to the literature on executive functioning, it is the process of working memory that appears to be implicated in the observed memory deficits in FASD. The present study will be the first to directly investigate this by examining both executive functioning and working memory with the expectation that this will produce some similar results thus strengthening the position that both are influenced by a common underlying mechanism. In doing this, this study will be the first to assess FASD using the Children's Memory Scale (CMS) to evaluate memory deficits, and the first to directly compare the memory and executive functioning using the CMS and the Children's Category Test. In addition, these areas of functioning will be studied for a group of children diagnosed within the standardized system of diagnosis established at the University of Washington (Astley & Clarren, 1997), thus ensuring accurate identification as well as allowing for clear comparison in future research.

Present Research

In the present study, the intent was to explore the possibility that a common underlying mechanism was responsible for deficits observed in both memory and executive functioning for children with FASD. If performance in these two areas is influenced by this single mechanism, it is plausible that performance in one area would predict performance in another area in which that same mechanism is active. Therefore, four predictions were tested to explore the possible role that this common element may play for children with FASD within their memory and executive functioning systems, (1) that there would be a positive correlation between measures evaluating memory and executive functioning, (2) that performance on three elements of memory, learning, attention and visual memory would predict performance on a measure of executive functioning, (3) that verbal memory would not predict performance on executive functioning, and (4) that ability as measured using an intelligence test would have no ability to predict performance on either a memory measure or a measure of executive functioning. To test these predictions, standard scores across measures of intelligence (Weschler Intelligence Scales for Children, 3rd Edition; WISC-III), executive functioning (Children's Category Test; CCT), and memory (Children's Memory Scales; CMS) were compared to determine what type of relationship exists between these three areas.

This research will advance practitioners' knowledge of the fundamental deficits present in children with FASD. While a large amount of variability is described for the deficits present in children with FASD, some similarities also seem evident. In particular, there appears to be some common ground between the research focused on Executive Functioning, and that focused on memory. For instance, planning skills, selective inhibition, and mental flexibility are concerns that have emerged in both bodies of research. Kodtuwakku et al. (1995) first identified this connection when they proposed that an underlying mechanism may be responsible for the deficits observed in each area. As referred to previously, they suggested that the Central Executive, a component of working memory, may be a key component in the deficits observed in FASD. Considering this approach, the research seems congruent. Encoding deficits, described with increasing consistency, could be attributed to deficits in this area. If the deficit is with the ability to appropriately direct or handle information, or even consistently attend appropriately, then naturally the ability to learn the information would be affected. Moreover, if attentional difficulties described in FASD are actually difficulties selectively attending, or focusing attention because of the working memory, than the inconsistencies in the attentional research thus far, which have focused on ADHD as a comparison, may be working from the wrong premise (e.g. Coles, Platzman, Raskind-Hood, Brown, Falek & Smith, 1997; Fried et al., 1992; Mattson & Jernigan, 1994; Nanson & Hiscock, 1990). In fact these attentional problems may be better understood from a memory model rather than an arousal model.

Could a common underlying mechanism be responsible for the types of deficits observed in these different areas of functioning in children with FASD? To continue exploring the possibility that this underlying mechanism may exist, comparison between memory and executive functioning measures is necessary. If the same mechanism is implicated in both functions, then it is possible that a comparison between two measures will reveal a positive correlation between the two. Such a correlation will lend support to the existence of a common underlying mechanism, and will alert researchers and encourage continued exploration into such a mechanism. In addition researchers may begin to define the nature of that mechanism by continuing to link the brain systems affected by alcohol to the functional deficits that are created by this damage. In this way, researchers and clinicians can identify the root of the deficits present in FASD in functional terms, and thus better understand the way in which cognitive systems are implicated by these deficits, ultimately culminating in the symptoms observed in children with FASD. Ultimately, this knowledge can lead to interventions that target the root of the problem and in doing so are able to best address the complexity of FASD.

As such the following predictions will be tested in this research. First, the CCT is expected to be positively correlated with the general memory score on the CMS. This would suggest that the two measures have some ability to predict performance on the other, possibly due to a common underlying mechanism that both measures are tapping into. While a relationship has been identified between working memory and executive functioning in previous research, the small number of participants prevented generalization of results (Kodituwakku et al., 1995; n=10) and further research was encouraged. While both memory and executive functioning in children with FASD have been independently studied since, they have not been directly compared in search of a common element of impairment between them. Moreover, the CMS and CCT have never been used together with children with FASD. Also important to this study is the use of children diagnosed at a later age in life (ages 9-16 years), as well as use of the diagnostic criteria described by researchers at the University of Washington (Astley & Clarren, 1997). Combining these unique features, this study intends to re-evaluate the possibility of a common underlying mechanism that may be affected in FASD, using a larger sample, through comparison of scores on the CCT and the CMS.

Second, a positive correlation is also expected between the CCT and the learning, attention/concentration, and visual memory indexes on the CMS.

Constructs of learning and attention are described for both memory and executive functioning. Consequently, it is hypothesized that the children's performance on learning and attention tasks, on the CMS, will be able to predict performance on the CCT. While similar to question one, this question narrows the exploration to those areas on the memory measure that correspond to the specific areas previously theorized to be related (Kodituwakku et al., 1995). This is also consistent with correlations reported between the CMS and the CCT (Cohen, 1997) in normally-functioning children, suggesting a similar construct exists between the two areas of functioning. In addition, in other independent research within these areas of executive functioning, and learning, researchers have described attentional and learning deficits for children with FASD as contributing to the deficits observed in both areas (e.g., executive functioning: Mattson, Goodman, Caine, Delis, &Riley, 1999; memory: Mattson & Roebuck, 2002). This then bears the question as to whether the deficits measured in executive functioning and memory would be better accounted for by attentional or learning deficits that may form the foundation of both functions. Visual memory (as measured on the CMS) is also expected to have some ability to predict performance on the CCT due to the visual nature of both tasks.

Third, no significant relationship is expected between the CCT and the auditory/verbal memory index. While attention and learning are key factors in both verbal memory and EF, other factors including comprehension skills and memory strategies will likely play a unique role in verbal memory. For instance, difficulties in comprehension reported for children with FASD (e.g., Streissguth, 1997) may interfere with their performance on verbal memory items in which comprehension is an asset (e.g., memory for stories). A positive correlation is reported for this relationship in healthy children (Cohen, 1997), however it is noted that the strategies employed by the children likely played a role in this relationship. The factors affecting the tendency of FASD children to use similar strategies are many and varied, and likely go beyond the role of Executive Functioning and Working memory (e.g., educational background, cognitive ability, and language skills).

Fourth, no significant relationship is expected for comparison between either the CMS and the WISC III, or the CCT and the WISC III. Intelligence has been described as extremely variable in FASD and has generally been found to have no ability to predict performance on other measures, though it is still necessary to rule out intelligence as the common factor (Mattson & Riley, 1998). Similar results are expected here, again indicating that while it may be affected by deficits in other areas, intelligence is not the underlying mechanism at work.

Methods

Participants

Thirty children with FASD participated in the study. All participants were born to mothers who abused alcohol during pregnancy, as determined through maternal reports, medical records, or information provided through Child Welfare. These children were diagnosed as having FASD using criteria based on the 4-Digit Diagnostic Code (Astley & Clarren, 1997), an important component unique to the current study, and recruited through a private practice that specializes in FASD diagnosis in conjunction with a community-based FASD diagnostic team. In addition, the following criteria were used in the selection of participants: fluency in English, no significant hearing or visual impairments, a minimum IQ score of 70 (Borderline range or above) as measured on the

Wechsler Intelligence Scale for Children (WISC-III), and no current diagnoses of severe psychiatric disorders (e.g. psychosis). A significant amount of screening was required in order to meet these criteria, and consequently fourteen months were required to compile the current sample of 30 children from the 64 that were screened. Thirty children tested were found to have IQ scores below 70, and consequently were not included in this study. In addition 4 children withdrew their assent or were not compliant with testing procedures and were therefore not included in the study. The participants had a mean age of 12 years 10 months, and ranged in age from 9-0 to 16-11. There were 14 females and 16 males. Their scores on the WISC – III ranged from 71 to 107, with a mean standard score of 84.13.

Measures

Four Digit Diagnostic Code. All children in this research were diagnosed using the standardized criteria developed and recommended by researchers at the University of Washington (Astley & Clarren, 1997). Briefly, this system documents the magnitude of expression of the four key components of the syndrome, specifically: (1) growth impairment, (2) the FAS facial phenotype, (3) evidence of brain damage, and (4) prenatal alcohol exposure, on separate four-point Likert Scales (Astley & Clarren, 2000). A rank of "1" on any scale means a finding within the normal range. A "4" on any scale indicates a finding that corresponds with accepted cases of FAS. A score of "2" or "3" specifically defines intermediate steps between typical and atypical presentation of FAS characteristics. These scales do not necessarily measure increasing severity, but rather they are scales of greater confidence that FAS is present, and as such, a diagnosis of FAS requires ranks of 3 or 4 in all categories. There are, however, many other possible alcohol-related diagnoses provided depending on the code obtained, in which case, there is much more room for consideration of the entire spectrum of this disorder.

All of the children in this study received a brain score of '3' indicating the presence of probable brain dysfunction based on performance of greater than two standard deviations below the norm in intelligence, achievement, adaptation, neuropsychological measures, and language. Also required were alcohol exposure scores of '3' indicating that while alcohol use during pregnancy is confirmed the exact amounts consumed are unknown. Together, these two scores are sufficient to indicate the presence of Alcohol Related Neurodevelopmental Disorder (ARND) in all children included in this study. Scores on growth and facial features, while utilized in the diagnostic process, were not considered as criteria for the purposes of this study, although no children had scores of '4' on either scale indicating that no children in this study presented with 'full FAS.'

Assessment of Executive Functioning, Children's Category Test (Boll, 1993). The Children's Category Test (CCT; Boll) was designed to assess cognitive processes required for successful academic achievement by evaluating a child/adolescent's ability to learn, solve problems, and develop, test, and modify hypotheses (Boll). Designed by Thomas Boll, the CCT is an adaptation of the Category Test that is part of the Halstead-Reitan Neuropsychological Test Battery. The CCT has two levels: level I for children 5 to 8-11 years old, and level II for children 9 to 16-11 years old. Given the ages of the children, only level II was used in the research. On this measure the child is asked to decide what number, from one to four, is represented by the symbols shown to them. The child is presented with five groups composed of these symbolic figures, and each group is consistent uses one theme to illustrate the four numbers. Using trial and error (they are informed of right and wrong answers), the children are encouraged to figure out the theme depicted within each group, which will then enable them to correctly identify the number represented by each symbol within that group. Scores are calculated by adding the number of errors made and then using the normative data provided to convert the raw score into a *t*-score.

The internal consistency of the CCT, level II, is very good, ranging from .81 to .89, with an average of .86 (Boll, 1993). The test-retest reliability scores average .75 which is moderate to good. The reduction in the test-retest reliability correlation may be attributed to the fact that the test is about learning, and therefore you will likely see less errors the second time the test is administered as there is more opportunity for learning to take place.

Evaluation of construct validity entailed comparison with traditional measures of intelligence, and memory (Boll, 1993). As the CCT is predicted to measure learning, it should not be related to prior learning, and so a low correlation with the WISC-III was expected and found ($r = .22$). Comparison with achievement measures also produced very low correlations. To assess criterion validity the CCT was compared to the Category Test. This comparison produced an adequate correlation of .72.

Assessment of Memory, Children's Memory Scale (Cohen, 1997). To assess various components of memory, the Children's Memory Scale (Cohen, 1997) was used. The Children's Memory Scale (CMS) is a nationally standardized measure normed on 1,000 normally functioning children aged 5 through 16. It is comprised of nine subtests which assess functioning in each of four domains: auditory/verbal learning and memory (verbal), visual/nonverbal learning and memory (visual), attention/concentration (Cohen), and learning. The nine subtests include six core subtests: (1) dot locations, (2) faces, (3) stories, (4) word pairs, (5) numbers, (6) sequences. The remaining three subtests are considered supplementary: (7) family pictures, (8) word lists, and (9) picture locations. Each domain is assessed through two core subtests and one supplemental subtest. For each subtest normative scores (x=10, SD=3) are provided for evaluating specific abilities, and eight index scores (x=100, SD=15) are derived from the core subtest scores. In all the entire measure takes between 45 minutes and one hour to administer.

Reliability coefficients were obtained for each subtest on the CMS at each age (ranging from 5 - 16 years). The coefficients range from a low of .61 for nine-year olds on that subtest which measures learning of dot locations, to a high of .94 for 13 and 14 year olds on that subtest which measures learning word pairs. Reliabilities were also computed for the average across the ten age-groupings. In this case the range was from .91 for learning word pairs to .71 for the immediate recall of faces. Reliability coefficients were also computed for the eight index scores, for both individual ages, as well as across age-groupings. Reliability of the index scores, as averaged for the across age-groupings ranged from .76 for both visual immediate and visual delayed memory, to .91 for general memory. An interscorer reliability study was also designed and implemented and the data provided shows that the interrater consistency is very high (Cohen, 1997).

Correlation to other key measures was also reported for the CMS. In a normal population the CCT is found to be moderately and positively correlated with the General Memory (r = .59). In addition positive correlations were reported for the CCT and two indexes on the CMS: auditory/verbal memory, and Learning indexes. Finally, in healthy children, memory functioning as measured by the CMS was found to be positively correlated to intelligence ($r = .58$).

CMS scores were also assessed for their stability over time (Cohen, 1997). Drawing from three age bands, 5-8 years, 9-12 years, and 13-16 years, 125 children were retested. The mean test/retest interval was 59.6 days. According to the data provided, CMS indexes generally possess adequate stability across time and age groups. Two areas however, visual immediate and delayed indexes, were reportedly lower than would be expected. Cohen suggests that this could be due in part to the nature of memory testing (e.g. procedural learning effects and re-exposure to learned material) and psychometric considerations.

Construct validity was assessed through subtest and index intercorrelations (Cohen, 1997). Results of the correlation analysis indicate moderate to high positive relationships between immediate and delayed recall measures within each subtest. The General Memory Index exhibits a moderate to high correlation with other memory indexes (ranging from .68 to . 79). Correlations are higher within domains than between domains for all indexes.

Assessment of Cognitive Functioning, Weschler Intelligence Scale for Child/adolescent – Third Edition (WISC-III; Weschler, 1991*)*. The WISC-III is used to evaluate cognitive functioning through the administration of as many as thirteen subtest. These subtests make up a Verbal Scale and a Performance Scale as two important dimensions of cognitive ability. The Verbal Scale includes five subtests: Information, Similarities, Arithmetic, Vocabulary, and Comprehension. The Performance scale also includes five subtests: Picture Completion, Coding, Picture Arrangement, Block Design, and Object Assembly. Three supplementary subtests are also included: Digit Span, Symbol Search, and Mazes. The WISC-III was standardized on 2,200 children, using sampling methodology that has been described as excellent (Sattler, 2001). In addition, the WISC-III is described as having outstanding reliability, with internal consistency reliability coefficients of .89 and above, and good test-retest correlations being reported, indicating both short-term and long-term stability of WISC-III scores (Sattler; Weschler). In addition, good construct validity is described (Sattler; Weschler). All core subtests were administered in order to obtain a full scale standard score (*M* = 100, *SD* = 15), which was used both for screening and analysis.

Procedure

Children and adolescents were tested as a part of a neuropsychological assessment. The CMS, CCT, and WISC -III were administered either by the author or a trained examiner using the standardized instructions. Testing lasted between two and three hours for each child. All testing procedures were approved by the Research Ethics Board at the University of Alberta, and informed consent and assent were obtained from parents and children, respectively, before beginning their involvement in this study (see Appendix A). All guardians and children were informed of their right to withdraw from the study at any time if they so chose.

Results

A wide range of cognitive and behavioural deficits has been attributed to the effects of prenatal alcohol exposure (Mattson & Riley, 1998). While some deficits seem to be more consistently present in these children, the relationship between areas of functioning is not always predictable. Likely because of the tremendous variability in factors affecting alcohol exposure, such as amount, frequency, timing, and protective factors, there is a wide range of effects possible in FASD. And the presentation of these factors may not always make sense within traditional frameworks of assessment. The current study was conducted to explore whether there was any correlation between three measures of functioning, across three different domains of functioning: memory, cognitive ability, and executive functioning for children with FASD. It was predicted that, (1) a positive correlation would be found for scores obtained on the general memory score of the CMS and the CCT, (2) a positive correlation would be found for scores obtained on the visual, attention/concentration, and learning indexes of the CMS, and the CCT, (3) that no significant relationship would be found for scores obtained on the verbal index of the CMS and the CCT, and (4) that no significant relationship would be found for general memory on the CMS, the CCT, and the full scale score on the WISC-III.

Pearson correlations were used to assess the relationship between level of functioning as measured by the CCT, cognitive ability as measured by the WISC-III, and degree of memory as measured by the general memory score on the CMS. Criterion for classifying correlations was as follows, a low correlation is < .3, a moderate correlation is between .3 and .7, and a high correlation is > .7 (Glass & Hopkins, 1996). Interestingly, there was a high degree of variability within the data collected. On the WISC-III the average standard score obtained was 84.13, and the range was 36 points. The CCT revealed similar diversity. The test average was 50.83, with a range of 34 points. Even greater diversity was found on the CMS. On this measure the mean was 86.00, with a range of 51 points. While a high degree of variability was expected, the extent to which it was observed was surprising. Descriptive results are presented in Table 4-1, and results from the Pearson correlations are presented in Table 4-2.

Table 4-1

Level of Functioning on the CMS, CCT, and WISC III

Test	*n*	*M*	*SD*	*Minimum*	*Maximum*
WISC III	30	84.13	9.18	71	107
CCT	30	50.83	8.80	32	66
CMS	30	86.00	12.13	56	107
Visual Index Immed.	30	91.93	12.49	57	115
Visual Index Delayed	30	92.37	15.34	60	115
Verbal Index Immed.	30	85.90	12.65	54	109
Verbal Index Delayed	30	89.90	12.00	57	115
Learning Index	30	83.30	12.04	57	112
Attention/Concen.	30	85.43	17.66	57	131

Table 4-2

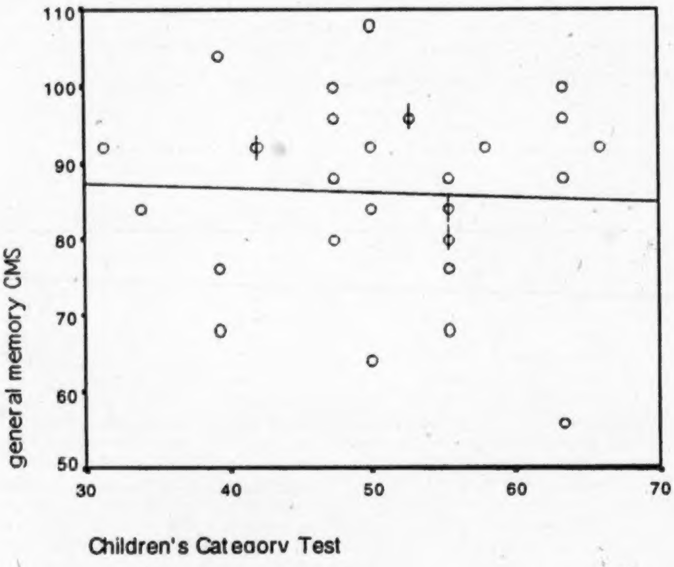
Intercorrelations Between Level of Functioning on the CMS, CCT, and WISC III (n=30)

Test	1	2	3	4	5	6	7	8	9	10
1. WISC III	--	.30*	.28	.39*	.06	.14	.18	.13	.35*	.05
2. CCT		--	-.05	.08	.17	-.25	-.18	-.13	-.11	-.17
3. CMS			--	.68*	.59*	.60*	.67*	.56*	.25	-.21
4. Visual Index Immed.				--	.58*	-.02	.12	.50*	.25	.04
5. Visual Index Delayed					--	-.15	-.10	.12	-.23	-.17
6. Verbal Index Immed.						--	.82*	.37*	.23	-.29
7. Verbal Index Delayed							--	.48*	.45*	-.13
8. Learning Index								--	.41*	-.06
9. Attention/Concen.									--	.18
10. Age										--

*$p < .05$ (two-tailed)

Also unexpected was the very low correlation between Executive Functioning and Memory. The first prediction, that the CMS and CCT would be positively correlated, was examined using a Pearson Correlation. Comparison between general memory on the CMS and the CCT produced a correlation of -.05, indicating no relationship between these variables (see figure 4-1). This is particularly interesting as it is in contrast to reports of a moderate, positive correlation between these two ($r = .59$; Cohen, 1997) in a general population.

Figure 4-1 – correlation between CCT and CMS



To test the second and third predictions, regression analyses were conducted for the CCT and, verbal, visual, attention/concentration, and learning indexes of the CMS. As time was not a factor in this prediction, the immediate and delayed indexes for verbal and visual memory were combined resulting in two variables: verbal (immediate and delayed) and visual (immediate and delayed). The dependant variable for this analysis was executive functioning as measured by the CCT, and the four independent variables were the verbal, visual, attention/concentration, and learning indexes of the CMS. No significant relationship was identified for any of the variables. The results of this analysis are presented on Table 4-3.

Table 4-3

Summary of Simultaneous Regression Analysis for Variables Predicting Performance on the CCT (n=30)

Model

R	*R Square*	*Adjusted R Square*	*Std. Error of the Estimate*
.28	.08	-.07	9.10

Coefficients

Variable	*B*	*SE B*	*β*	*Power*	*p*
Visual Memory	.12	.15	.18	.09	.41
Verbal Memory	-.12	.17	-.17	.09	.47
Learning	-.09	.18	-.12	.09	.64
Attention/Concentration	.002	.11	.004	.09	.99

The fourth prediction, in which no significant relationship was expected between the CMS, the CCT, and the WISC III, was tested. Remarkably, the strongest relationship between overall measures was found for WISC III scores and the Children's Category Test, falling into the low end of the moderate range. This correlation was found to be .30, indicating that 9 percent of variance is accounted for between these two measures (see figure 2). This was unexpected given the low correlations typically reported between these instruments (r = .14 -

.27; Boll, 1993), though perhaps not far from the expectation that the performance of FASD children on this measures would not fit within generally expected paradigms. As predicted, a low correlation of .28 was identified for the relationship between the CMS and the WISC-III, in contrast to the moderate positive correlation reported for the general population (*r* = .58; Weschler, 1991).

To further examine this relationship, a regression analysis was conducted to evaluate the relationship between WISC-III scores and the CCT and CMS variables. The dependant variable was the WISC-III and the independent variables were the CCT and the CMS. As was predicted a significant relationship was not identified, indicating that the CMS and the CCT are not significant predictors of performance on the WISC-III for children with FASD. These results are presented below on Table 4-4.

Table 4-4

Summary of Simultaneous Regression Analysis for Variables Predicting Performance on the WISC-III (n=30)

Model

R	*R Square*	*Adjusted R Square*	*Std. Error of the Estimate*
.43	.18	.12	8.61

Coefficients

Variable	*B*	*SE B*	*β*	*Power*	*p*
CCT	.33	.18	.32	.22	.08
CMS	.23	.13	.3	.22	.10

Ad Hoc analysis of the relationship between CMS indexes and the WISC-III revealed two moderate correlations with the visual immediate index (*r* = .39) and the attention/concentration index on the CMS ($r = .35$). This indicates that 15 and 13 percent of variance, respectively, is accounted for by these two measures, in these two areas. The relationship between age and the CMS, WISC-III, and CCT was also examined. While only low correlations were identified, it was interesting to note that most of the correlations observed were negative. This may suggest that standard scores declined somewhat with age. This is an area in which further study is recommended.

Discussion

Memory and Executive Functioning

The hypotheses made in the current study reflect recent studies in which researchers have stated that while there is not a clear pattern to the deficits present in children with FASD, some consistent themes have emerged between executive functioning and memory (e.g. Mattson & Riley, 1998). It has been postulated that an underlying mechanism is responsible for observed deficits in both areas (e.g. Kodituwakku et al., 1995). As such, the first three predictions in the present study attempted to explore this possible relationship through comparison of performance by children with FASD on the CMS and CCT. The results found here, however, do not support this supposition as no strong relationship was identified for memory and executive functioning.

Though no additional evidence was found for a common mechanism between these areas, it is possible that this may be due to limitations posed by the tests used rather than the absence of a relationship. In particular scores on the CCT were much higher than expected and even when the children had great difficulty on sections of the test, these problems were not reflected in their standard scores, which were often within a standard deviation of the average score. Only when the children were very clearly impaired, did these scores reflect the observed problems. Of interest, it was noted that when the children were unable to figure out a specific subtest it was observed that they tended to respond very quickly to the items with no apparent effort to apply a consistent strategy to solve the problem. Moreover, the children would often 'guess' a number outside of the four they had to choose from suggesting they struggled to work within the rules of the test. This pattern of performance is consistent with research in this area in which deficits in response-inhibition have been linked to difficulties with judgement and planning described in parent reports (Kodituwakku et al., 1995; Mattson, Goodman, Caine, Delis, Riley, 1999). Thus, it seems that while problems were observed in this area, these concerns were not reflected in the scores obtained. This potential lack of test sensitivity is also reflected in the low statistical power reported for the regression analyses, indicating that the ability to pick up an existing relationship was not strong. Two areas of further research could help to clarify this issue. The first would involve administering different measures of executive functioning to children with FASD and a control group in order to determine which measure best distinguishes the two groups. This measure could then be used in the second area of future research in which comparison to performance on this new measure and the CMS could be conducted.

In the fourth prediction for this study, it was expected that there would be very little correlation between executive functioning and cognitive ability indicating that performance on the CCT was independent of cognitive ability. Surprisingly a moderate relationship was identified between executive functioning and cognitive ability, though further analysis did not find this relationship to be statistically significant. Nonetheless, this marginal overlap may indicate that while current ability to learn is not dependent on past learning, that ability to learn and problem solve may equip children with FASD to adapt to cognitive demands more effectively. Conversely, if children possess good cognitive ability they may be better able to solve the problems presented by the CCT. While this finding was unexpected given previous research which has found no significant relationship between executive functioning and IQ (Mattson, Goodman, Caine, Delis, & Riley, 1999), this could reflect the limitations of the CCT to fully explore all components of executive function. Further research in this area may produce greater insight into the relationship of executive functioning and cognitive ability with this population.

Analysis of the role of age revealed another area in which further study is warranted. Specifically, a slight trend towards lower standardized scores in memory and executive functioning with increased age suggests the possible presence of a depressed developmental curve in FASD. Current longitudinal studies being conducted at the University of Washington will hopefully help to define the unique developmental processes in FASD.

Memory and Cognitive Functioning

Additional exploration also revealed moderate correlations between the visual immediate and attention/concentration indexes of the CMS and the WISC-III. This may indicate that both the ability to visually process information as well as the ability to mentally manipulate information through sustained and focused attention are important for performance on the WISC-III. This conclusion is consistent with a study by Streissguth et al. (1990) in which they found Digit Span and Arithmetic to be most highly related to prenatal alcohol exposure, and suggested that IQ deficits observed on intelligence tests may be attributable to other factors. Moreover, those same functions could then also be implicated as underlying mechanisms responsible for some of the cognitive deficits observed in FASD. Clearly, however, these conclusions are exploratory in nature, primarily highlighting possible areas of future research.

Conclusions and Future Directions

Overall, the relationships that are typically described between these instruments within the general population were not present for children with FASD. Thus, these unusual results challenge some of the typical assumptions made in conducting an assessment, and support the position that this is a truly unique population requiring special consideration. This is not to say that the research eliminates the possibility that patterns exist within this population, but rather it suggests that the absence of any clear or dominant pattern appears to be a fundamental characteristic of FASD.

In terms of a practical interpretation of these findings, useful for those conducting assessments, these results emphasize the need to move away from using generalizations. Children and adolescents with FASD need to be considered individually, using comprehensive measures that permit thorough evaluation of all areas of functioning. In addition, ability in one area cannot be assumed to mean ability in another area. Comparison of these areas revealed individuals who obtained WISC-III and CMS scores with a thirty-point separation. Clearly, if an assessment is not sufficiently comprehensive, then deficits may not be properly identified, and false conclusions could be drawn. So, while there is no one test or one pattern that can be used to identify children with FASD, use of these instrument remain clinically useful for detecting cognitive or functional deficits that may be present in children with FASD. Moreover, the diversity identified in these three measures suggests that all are useful components within a thorough assessment of deficits present in FASD, as they all seem to identify different factors. Continued research in this area to further develop information on clinically useful test batteries is needed. In addition to individualizing assessment procedures to account for this wide range of variability within FASD, changes to traditional school and mental health categories of impairment are required so that the unique nature of FASD does not preclude access to necessary funding and support. For example, as this study has demonstrated, an Average intelligence may be accompanied by significant problems with memory, which would contribute to difficulties in educational as well as daily functioning.

While the ultimate goal is prevention of FASD, there is a realistic need for accurate identification and appropriate intervention for those children with FASD. To do this there must be an awareness of the types of deficits that may be present within this population, such as memory and executive functioning, as well as the fact that there may be no pattern to these deficits. As the present research shows, there is a high level of variability in individual presentation of deficits, making widely generalizable predictions difficult to establish. Nonetheless, the provision of a standardized method of diagnosis, created by researchers at the University of Washington, permits a thorough assessment that encompasses the diverse presentation of FASD (for more information, see Astley & Clarren, 2000). Given the extent of the complexity of this disorder, as briefly demonstrated here, investigation and training in this way seems necessary. If each child is to receive the best care possible, through identification of problem areas and specific intervention geared towards those areas of deficit, then a comprehensive approach is essential. Not only will this provide immediate and appropriate support for the child, but in doing so may also help to prevent some of the secondary disabilities reported in FASD.

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V. Conclusion

The goal of studying FASD is to better understand the types of deficits evident in children with FASD, and in turn to more effectively diagnosis and intervene appropriately. These basic goals are followed by more specific questions: Is there a pattern to the deficits present in FASD that may suggest that specific mechanisms are responsible for many different observed dysfunctions? What is the best way to respond to the deficits present in FASD?

Each of these questions poses unique challenges to researchers and clinicians, which are complicated by the lack of consistent understanding by practitioners potentially working with this population. Streissguth (1997) has stated that "many professionals do not yet have the nomenclature to fully understand the syndrome and its implications or really 'hear' what parents are saying" (p.7). She continues to explain that while research has grown in recent years there is still a lot of confusion about FASD, and that there is a gap between the scientific knowledge and general clinical information. Six years later, her comments still apply. Despite efforts to spread the knowledge of FASD, many remain unaware of the unique presentation of this disorder. Consequently, research in this area must continue to include studies and reviews relevant to both clinicians and scientists in an effort to bridge this gap.

One way to do this is through use of theoretical models that facilitate the interpretation of data that is compiled by defining a functional framework. A second way is to use comprehensive clinical tests that provide functional information for clinicians. It is particularly helpful when the measure used corresponds with the theoretical model as it provides a context within which the results can be applied. Not only does the combination of these two elements contribute to understanding and clinical application, but also enables scientists to ensure that they are testing and comparing similar things. This kind of comprehensive assessment strategy, tied to a standard of diagnosis such as the four digit diagnostic code, will provide the groundwork for a consistent and optimal system of diagnosis and intervention.

In FASD prenatal alcohol exposure has resulted in a myriad of problems, which often result in the inability to integrate past experience into current knowledge, which is a source of great frustration for those with FAS and a cause of dysfunctional and maladaptive behaviours (Streissguth, 1997). Considering this difficulty effectively learning from past experience, it is easy to see how behavioural problems are often the most overt presentation of FASD. Unfortunately, if these behaviours are misunderstood, or misdiagnosed, problems will persist. Consequently, the first step to addressing maladaptive behaviour in FASD is to better understand the mechanisms contributing to any observed problems. Thus, the methodological questions we should ask are: (1) What kinds of patterns can be identified in the deficits present in FASD?, (2) How can we best respond to these patterns in terms of expectations and realistic goals?, and (3) What types of intervention are most appropriate for this population?

Studies conducted as part of this dissertation respond to the first question. In what follows I have first summarized the main findings and then discuss the implications and limitations of each. Each section includes some suggestions for future research.

A Review of the Evolution of FASD

In chapter II, I suggested that valid and reliable diagnostic processes are necessary for FASD. Through a review of the evolution of diagnosis in FASD the importance of accurate diagnosis is underscored. Some explanation is also offered to the unique challenge that this disorder has posed to both clinicians and scientists in terms of both definition and description, given the variety of deficits that have been attributed to FASD (Mattson & Riley, 1998; Mattson & Roebuck, 2002). Current strategies of diagnosis were introduced in the expectation that this knowledge may guide clinicians in their practice with this unique population (e.g., Astley & Clarren, 2000).

Patterns of Acquisition and Retention of Verbal and Nonverbal Information in Children with Fetal Alcohol Spectrum Disorders

In this study, Multivariate Analysis of Variance was used to explore the relationship between different memory functions. Specifically, this included: 1) visual memory and its components of object and spatial memory; 2) two elements of verbal memory; and 3) processes of learning or encoding information. This was accomplished by using a model of working memory as a framework (Baddeley & Hitch, 1974) and a comprehensive memory measure (Cohen, 1997) that is consistent with this theoretical model.

Results indicated statistically significant differences between both aspects of verbal memory. Verbal memory was operationalized in two ways; memory for stories and memory for word pairs. In this study, memory for word pairs was significantly more impaired than that for stories. It is offered that this difference may be attributed in part to the different function of perseverative errors between the two tasks. In the first, word pairs, perseverative errors compromised accuracy and presented as an ineffective strategy in which the same word would be offered until a match was found. In stories, however, the children tended to "add in" information that made sense to then. In this way they seemed to perseverate on a theme. As such, even if they could not remember the details they seemed to generate them as they created their own story line. The children were not penalized for lack of accuracy and so this tactic actually seemed beneficial for this particular task.

It was also proposed that the children's performance with stories could indicate that the memory systems between the two functions are different: word pairs involve recitation while stories may use chunking strategies that link with experience. Implications for the particular deficits observed for word pairs were described. Specifically, research connecting this memory function to vocabulary acquisition was discussed, and future research into this possible relationship is recommended. Further research into the different components of verbal memory and the implications of specific deficits among these components in FASD was also identified as an area of need.

Differences were also identified between delayed and immediate verbal memory. This finding is consistent with previous research in which learning or encoding has been identified as the greater problem for FASD children rather than the process of accurately retaining and retrieving information (Kaemingk, Mulvaney, Halverson, 2003; Mattson & Roebuck, 2002). No significant differences were identified within visual memory, though the complexity of this system and the differences in the literature are explored. Consequently this area was identified as one needing more research to better understand differences in visual memory in general and in FASD in particular.

This study was limited in part by the large amount of variability within the sample. This variability was described, along with implications for such diversity within the FASD population. Another limitation in the current study was the number of participants, thirty adolescents and children in total. While the sample size was larger than is typically seen within research in this area (e.g. typically n=>20), caution must still be exercised when deriving generalizations from this study, and for this reason evaluation of statistical significance was conservative. At the same time, it is possible that a larger sample could mask the unique nature of this population. In other words, large numbers may produce statistically significant results, though the clinical significance of such results may be suspect. Nonetheless, continued study with this population, using both large and small groups will contribute to a comprehensive picture of this population in which the ability to generalize will likely always be limited due to the diverse presentation of this disorder. Future researchers in this area may also want to examine other memory models to determine whether the deficits present in FASD could be better understood using a different framework.

Interrelationship of Neuropsychological Deficits in FASD

Correlational analysis was used to explore the possible existence of a common underlying mechanism that may play a fundamental role in executive functioning and memory. This analysis revealed no common mechanism through the measures used. Instead, the dominant feature was the large amount of variability both within and between participants. Consequently the need to individualize assessment for this population was emphasized. Moreover, the absence of deficits in one area may not indicate that related areas have no problems. As a result, an assessment with this population must be very comprehensive with examination into all areas of functioning. Only in this way will an accurate picture of a child or adolescent's deficits be obtained.

While three key areas of functioning were evaluated, many more need to be included in future studies. Academic performance, language skill, adaptive functioning, and behavioural components are just some of the areas that may benefit from similar comparisons in order to better understand their interrelationships within this population. Provision of a control group in future studies would also permit direct comparison between the differences in the relationships of these measures to one another for healthy versus FASD children. Moreover, in examining these areas of functioning, instrument sensitivity must always be considered. In this case, it is possible that the instrument used failed to effectively capture the types of EF deficits present in FASD. Comparison of instruments in this area, with this population, may provide insight into the best tools for use with this population.

The need to redefine public policy and educational funding in particular was suggested, in the light of the unusual presentation of deficits within this population. In addition, psychologists and other practitioners working with this population must be made aware of the unusual presentation of deficits within this population. Moreover, professionals who choose to work with children who may have FASD must stay abreast of current trends in diagnosis so that they can be consistent in their evaluation of the deficits present in FASD. Continued education and effective communication networks, while always important for professionals to maintain, are essential with this syndrome. Overall, consideration of the unique pattern of deficits within FASD is necessary for psychological, educational, and health professionals to appropriately meet the needs of this diverse population.

Future Directions of Research into FASD

Research into FASD has accelerated in the last five years. As we become more aware of the range and degree of deficits present in children with FASD, more questions are spawned. As such three general areas of exploration seem to be present. The first, described minimally here, relates to empirical studies using animals in an effort to describe the effects of alcohol on the structures of a developing brain. This vein of study is being enhanced by recent human studies in which current technology such as MRIs have enabled researchers to look at human brains for evidence of damage. A second focus of research is the one applied in the current study – examination of specific functional deficits in children with FASD. This body of research includes studies that attempt to explain the effects of this brain injury on daily functioning. A final, general area of study in this field explores interventions, and often includes practical suggestions for support in home or academic settings. Some qualitative research is also beginning to emerge in this area regarding the experience of living or working with children with FASD, or even having it, that is beginning to help in the process of putting a human face on this syndrome.

Clearly, these three areas of study are neither inclusive nor exclusive, but rather help to describe the many levels of study involved in better understanding FASD. In this study, areas of specific deficits have been examined, though clearly more questions have been raised than answered. This seems to be the nature of study into FASD, and simply means that as researchers we must be diligent in our research, and build new studies based on questions posed. Ultimately, the goal of research and practical work with FASD is to prevent as many children as possible from being exposed to alcohol prenatally. This research takes us closer to reaching this goal by allowing for accurate diagnosis of children with FASD, which in turn creates opportunities for preventative intervention through identification of families and individuals at-risk of drinking alcohol during pregnancy.

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Appendix A: Consent Form

Dear Parent(s) or Guardian(s),

I am a doctoral student at the University of Alberta in the department of Educational Psychology. Under the supervision of Dr. Christina Rinaldi, I am conducting a study to determine what types of memory processes are present in children diagnosed with either Fetal Alcohol Syndrome (FAS) or Alcohol Related Neurodevelopmental Disorder (ARND). Results from their performance on various tasks will be used to answer questions such as how memory is related to their academic performance, their social skills, and their cognitive ability.

Confidentiality of all information gathered from your child is assured. All data collected from the assessment will remain confidential and will be identified by a code number, and not by name, on the material associated with the study. Since participation is completely voluntary, your child may withdraw from the study at any time. Furthermore, in no way will your child's choice to remain in the study or to be removed have an impact on the assessment being completed. The assessment and the study will remain separate at all times.

I would greatly appreciate your child's participation in this study in order to help myself and others gain a better understanding of the memory processes associated with FAS and ARND. If you have any questions or concerns please do not hesitate to call Dr. Rinaldi at or myself at and we will return your call within forty-eight hours. Please complete the attached consent form. Thank you for your time and cooperation.

Sincerely,

Jacqueline Pei
Doctoral Student
University of Alberta

Christina Rinaldi
Assistant Professor
University of Alberta

Consent Form

Please return this permission form by the end of the testing day, REGARDLESS OF YOUR INTENTION TO PARTICIPATE.

Check the appropriate line:

_____I give permission______I do not give permission

for my son/daughter ___________________(PRINT child's name) to participate in Jacqueline Pei's (University of Alberta) study as part of her doctoral thesis work.

I understand that my child has the right to withdraw from the study at any time.

Signature parent/guardian

____

Please PRINT your name

____

Today's date_________________